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IN THEIR CONTRIBUTIONS

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

MARCH 1939

ADDRESS OF THE RETIRING PRESIDENT*

JAMES ALEXANDER MILLER

MY DUTIES and responsibilities upon this occasion are relatively unimportant and what I have to say will not detain us long from the main object of this annual meeting which is to usher in a new administration of the affairs of the Academy.

A very brief summary of our activities during the past year is, however, in order, but the details are contained in the various reports which have been presented to you this evening by title and which will be published in full in the regular annual report of the Academy. I trust they will receive from the entire membership the careful attention which they deserve.

The Trustees under the able chairmanship of Dr. Niles have managed the business affairs of the Academy with great devotion and skill. Our financial resources have been conserved with considerable success in the face of the great difficulties imposed by the general economic uncertainty and particularly and due largely to the efficient management of our Comptroller, Mr. Eberle, the Trustees have been able to place our investments in real estate mortgages upon an extraordinarily sound basis.

The general financial situation of the Academy has been the subject of great concern and the most careful study during the entire year. The

* Delivered January 5, 1939 at the Annual Meeting of the Academy

expanding activities and responsibilities of the Academy call for financial expenditures beyond our regular income. The Council and Trustees believe that these expenditures can not be materially curtailed without serious handicap to the program to which we are committed. It was determined therefore to make a systematic attempt to increase our income. Through a special committee, of which Dr. Krech is chairman, this effort has been very successful, but we still need additional funds, to obtain which the activities of this committee will be continued. I bespeak the even greater co-operation of the members of the Academy in order that this committee may attain the objects in mind.

Although it has been the opinion of the Council that all of the present activities of the Academy are important and essential, in view of the budgetary situation and also in accord with sound policy, the Council has authorized a very thorough-going study of all of the Academy activities. Through a large committee, divided into several subcommittees and under the general chairmanship of Dr. George Baehr, this analysis is now being made and its results will be available soon and will serve as an accurate basis upon which to formulate both the program and the budget for next year.

A quite unexpected and serious problem has recently been presented to the Trustees and Council by a request from the Federal Bureau of Internal Revenue to show cause why the Academy of Medicine should longer be exempt from taxation. At first it did not seem that this could be a serious request as the Academy ever since its original organization in 1847 has been exempt from all taxation as a quasi-public educational institution. This request soon developed, however, into a very serious and determined effort and it was only by the most detailed and skillful presentation of our case by our Director, Dr. Hartwell, and our legal counsel that our continued exemption from taxation was finally allowed. It is, however, important that all of our members should be made aware that the only basis for this favorable decision was the evidence presented of the large amount of education and of service that the Academy renders the community as a whole. It was the occasion for great gratification that a very large number of persons important in both public and private life in New York rallied to our support and offered the most enthusiastic testimony to the invaluable services that the Academy is rendering to the community. This incident should serve as a serious warning to future officers of the Academy and particularly to those members who have

entertained doubts concerning the wisdom of our policy to extend our services to the public as well as to our own membership.

As to the regular activities of the Academy I will comment very briefly and only to state that the work of the Library, of our Medical Education, of our Public Health Relations, and of the Medical Information Bureau have, under the direction of our various committees continued to operate upon the high level of efficiency which we have become accustomed to expect.

I may, however, take this occasion to emphasize the great success of the Committee on Education in the development of the Graduate Fortnight and the Friday Afternoon Lectures, and also I am personally particularly grateful for the inauguration of a new policy concerning the programs for the Stated Meetings. Largely through the initiative of a subcommittee of which Dr. Chace is chairman, these meetings have been transformed from a nightmare for the presiding officer to an occasion of great pleasure and educational profit. Also, I desire to record my great satisfaction in the action taken by the Academy tonight in transferring the Medical Information Bureau to the status of a regular standing committee of the Academy. This activity of the Academy is its most recent one and has furnished the occasion for deep searching of hearts on the part of the officers and some of our members. The Council has now, however, definitely taken the position that this service is not only a proper but also an important activity for the Academy to undertake and by so doing it has approved the judgment of our late Director, Dr. Linsly Williams, through whose efforts this Bureau was originally established.

To a considerable extent the work of this Bureau is still in a pioneer field and is too little known and appreciated by our membership as a whole. Its function may be briefly though inadequately summarized to consist in providing a clearing house for medical information for both physicians and laymen and especially to interpret new steps in the progress of medical science and to guide the method of its communication to the public, especially through the public press and the radio. In addition, the Bureau has organized a very successful annual series of Lectures to the Laity.

This program presents many new and at times knotty problems and to meet them the Committee and its executive secretary are aided by a special staff of experts in every field of medicine selected from the membership of the Academy.

By sanctioning this Bureau, the Academy has placed itself upon the side of general public education in medical matters and has discarded the theory that medical knowledge should be confined strictly to the medical profession. Mistakes and misinterpretation will inevitably occur in this field but it is not only my personal belief but also that of the Council that the program as a whole is most useful and is destined to be increasingly valuable by fostering better mutual understanding between physicians and the public which they serve. It is certainly in line with the spirit of the times and this was one of the public services of the Academy which favorably impressed the Bureau of Internal Revenue in its recent investigation of our activities. I hope that more of our members will get to know and understand this Bureau better.

While we are considering the relations of the Academy to the public, I would like briefly to touch upon another aspect of this question.

I have frequently been asked, "What is the position of the Academy of Medicine regarding the seething discussions which are going on concerning what is rather loosely termed, Socialized Medicine?"

As I understand it, it is the policy of the Academy not to participate actively in any discussion of the social and economic problems which concern primarily the organization of medical practice either in relation to society as a whole or to the State. This is the function of other duly authorized representatives of the medical profession.

As individuals, however, we are of course deeply concerned especially when the situation has developed that our profession is classed as a trades union and is indicted for maintaining a conspiracy in restraint of trade. We are indignant at the grossly unfair position into which these charges have forced us and we believe that this feeling is shared by most of the laity with whom we come in contact in our practice. As an Academy, however, we have no intention of jumping into the arena; but that a situation is developing which may have serious consequences, not only for the medical profession but also for the public which we serve, there can be no doubt.

It would seem to me that as an educational institution the Academy has an opportunity and a responsibility to be indirectly of great service in this matter. In the first place we know that really adequate medical service depends upon quality rather than quantity or methods of distribution. We know the rapid progress that has been and is being made both in medical knowledge and its practical application. We also know,

none better, that much inadequacy and imperfection as well as lack of knowledge still exist.

It would appear to be the duty of medical educational institutions like the Academy to redouble their efforts to remedy defects when possible and to maintain even higher standards in practice and in ethics than have been achieved in the past. The Academy can do this.

In the second place we can extend our efforts to educate the public so that it may better understand, not only what we have done and are doing, but also what we have thus far failed to do and why. The laity should know something of our difficulties and our problems as well as of our achievements, should appreciate the fact that real success in this difficult field cannot be obtained through legislation or State control, and finally should realize that our success or failure in overcoming our difficulties means even more to them than it does to us, and that to succeed we need their understanding as well as their help and co-operation.

In a democracy like ours the ultimate power is with the people and they, not the medical profession, will decide this question. From our vantage ground of more intimate knowledge we can but guide and help the people to reach a wise decision. Toward this end we should increase and extend rather than curtail our efforts to educate the public in medical matters. This also the Academy can do.

It would be impossible to relinquish the office of your President without expressing to you my deep appreciation of the services of the executive staff of the Academy and of the wonderful spirit which animates them throughout. It is they who make the Academy what it is and we as officers and members alike can never cease to be grateful to them and appreciative of their devotion.

Among such a fine group of people one hesitates to single out any one for especial mention. It, however, could surely not be considered invidious to point out that any executive staff takes its cue from its leader and in its leader, our Director, Dr. Hartwell, both the staff and the entire Academy have come to recognize a personality possessed of a rare combination of efficiency, good judgment, sympathetic understanding, political insight, and capacity for hard work. For five years the Academy has been his main, in fact it would seem as though it must have been his sole interest, so devoted has he constantly been to its welfare. That this has been so in spite of great physical handicaps and suffering, makes his

achievement truly remarkable and stamps him as a man of the highest courage as well as of high ideals. The Academy during its memorable history has enjoyed the devoted services of many notable officers, but I venture to assert that none will ever be regarded as having been more notable or more devoted than John A. Hartwell.

This now brings me to a very unwelcome task. I have to announce that Dr. Hartwell has presented his resignation as Director and that the Council has accepted it to take effect April 1, 1939. He will not really leave us for his influence will long, probably always, be with us; but we are sad at his going and all of us, officers, staff and members alike, bid him God speed and wish him many more years of renewed health, of service, and of happiness.

It was more than a year ago that Dr. Hartwell presented his resignation to the Council to take effect at its pleasure. With the inevitable prospect ahead that his successor must ere long be found, a special committee was appointed to canvass the situation and make recommendations to the Council. This committee has worked very hard and most faithfully. Its first objective was to find a comparatively young man who possessed the necessary qualifications. In that they were unsuccessful. They then explored the possibilities of the older age groups. In that they have been eminently successful and some of us, perhaps because we too are older, were glad, because we visioned the Academy as in need of a leader of ripe experience and seasoned judgment in these perplexing times.

The committee recommended to the Council the name of a man who stands very high in our profession as a clinician and as a teacher, who has had the experience of directing the clinical and research activities of a great hospital, who has served actively for many years upon various important committees and more recently as a Vice President of the Academy. He has demonstrated outstanding ability and also possesses personal qualities which will make for understanding co-operation and inspiring leadership. By invitation of the Council, our Director after April first will be Dr. Herbert B. Wilcox.

And now my task and my responsibilities are at an end. It remains only first to express to all of you, officers, staff and members, my deep appreciation of the great privilege that has been mine to have served as your President during the past two years. These years have greatly deepened my appreciation of and affection for this great institution. I would that I might have been able to be more effective and if perchance

anything of value has been accomplished, it has been only because of your splendid and devoted support for which I will always be in your debt.

And secondly and finally there remains to me a great pleasure and privilege, that is, to introduce to you your new President.

I have known him intimately for nearly fifty years. I have never known him to fail, either in friendship, in character or in devotion to the highest ideals of our profession. You may be assured that he will not fail you.

Your President! Dr. Malcolm Goodridge.

PRESIDENTIAL ADDRESS*

MALCOLM GOODRIDGE

I HAVE a very full understanding of the responsibility I have assumed, in accepting the great honor you have accorded me, in electing me your President.

I am acutely aware of my own inadequacy to meet the standards set by my illustrious predecessors in this office. However, humility is a solid foundation for virtue, and the very realization of my limitations is a challenge to my sense of pride; so I put all my fears behind me, and assure you I am entering upon my new duties with a spirit of determination and enthusiasm which, with your help, will overcome what at first seemed to be insurmountable handicaps.

I know eulogy is distasteful to Dr. Hartwell, because he has told me so; nevertheless, I cannot refrain from saying a word about him tonight. I have spent some time recently, studying the history of the Academy. All of the addresses of its Presidents, up to and including 1879, that are to be found, and many of those delivered since then, I have read, so I have acquired knowledge, not only of the authors of those addresses, but also of their opinions of their contemporaries.

I have known Dr. Hartwell as a man, as a friend, as a patient, and as a physician; I have known him also as President of the Academy and as its Director, so I speak with authority, when I tell you that in all its ninety-odd years of existence this Academy has never had a person connected with its organization who has given to it more of himself and given to better advantage.

He has taught me many things that cannot be learned from books. He has always had the capacity for making friends. In him you find kindness, truth, patience, and honor. He has been a wise and inspiring leader. We are going to miss him; but, long after our generation has passed on, his spirit will continue to live in the affairs of the Academy, more indestructible than its walls, and vastly more precious.

Nearly a hundred years ago, on the evening of November 18, 1846, at the Fourth Annual Dinner of the Society for the Relief of Widows and Orphans of Medical Men, held at a restaurant on Broadway, near

* Delivered January 5, 1939 at the Annual Meeting of the Academy

Prince Street, the idea of an Academy of Medicine was born. This was indeed the horse-and-buggy age, not only of medicine, but of science in general. Let me remind you that the population of what is now the Greater City of New York was in the neighborhood of six hundred thousand; the first railroad train drawn by a steam locomotive, the first Atlantic crossing of a steam-propelled ship, and the use of illuminating gas as a means of street-lighting had been introduced but a few years before the founding of the Academy, while the first commercial telegraph line in this country was operated between Washington and Baltimore in 1844. Oliver Wendell Holmes reported the infectious nature of puerperal sepsis in 1843; and three years later, but a single month before the birth of the Academy, William T. G. Morton demonstrated the administration of ether to a patient on whom John C. Warren was about to operate, in the amphitheatre of Massachusetts General Hospital.

The prime motive for the formation of the Academy was not at this time the advancement of medical science, but rather the formation of a code of ethics to regulate the professional conduct of its members. The original Constitution, adopted January 6, 1847, reads, under Article 2, "The objects of the Academy shall be:

"First. The separation of the Regular from Irregular Practitioners.

"Second. The association of the Profession Proper for purposes of mutual recognition and fellowship.

"Third. The promotion of the character, interests, and honor of the fraternity, by maintaining the union and harmony of the regular profession of the City and its vicinity, and aiming to elevate the standard of Medical Education.

"Fourth. The cultivation and advancement of the Science, by our united exertions for mutual improvement, and our contributions to Medical Literature."

So we see the Academy was originally started, not primarily as an institution for the benefit of mankind, but rather as an organization of one hundred twenty-one medical men whose chief aim was to draw the line sharply between regular practitioners and those whom they regarded as charlatans and humbugs.

The Standing Committees under the original Constitution were appointed by the President, and were a Committee on Admission, a Committee on Finance, a Committee on Medical Ethics, a Committee on Publication, and a Council of Appeal—in keeping with the objectives

stated in the original Constitution.

A year later, however, the order of the stated objects was changed, so that the cultivation and advancement of the Science, by united exertions for mutual improvement, and contributions to medical literature was placed in the leading position, while the separation of regular from irregular practitioners was relegated to third place.

In 1874, the "Promotion of Public Health" was added to the objects of the Academy.

Throughout the first twenty-eight years of its existence, the Academy of Medicine had no home. During most of this time, its small collection of books had no permanent resting place. On the occupancy of a building at 12 West 31 Street, in May, 1875, the Academy finally had a roof over its head. There were available for the Library four hundred volumes, and in June, 1875, a Committee on Library was added to the Standing Committees. From the time we occupied our first home up to the present time, we have been leaders in matters which affect public welfare and medical education. The Library increased rapidly in size until there were nine thousand volumes on its shelves in 1879, and twenty-five thousand in 1885.

The greatly beloved Abraham Jacobi had the vision, in 1885, to point out that the people would soon learn to rely on the knowledge and public spirit of the profession, and, as it depended on the Bar for legal advice, would consult the medical profession for sanitary necessities.

About this time, a bill was presented to the Legislature, ordering a Board of Examiners to institute State examinations, and a department to license practitioners of medicine; and the Academy of Medicine was the motivating force in finally securing such legislation.

Continued rapid expansion produced the need for larger quarters, and on November 20, 1890, a new building was opened, at 17 West 43 Street. Oliver Wendell Holmes is reputed to have said, in a letter of regret, written for this occasion, "Academies have been too often thought of as places of honorable retirement and dignified ease; roosts where Emeritus Professors and effete men of letters, once cocks of the walk, could sit in quiet rows while the fighting, the clucking and the crowing were going on beneath them . . . But the Academy which fulfils its true function is a working body. It deals with living subjects, it handles unsettled questions; . . . it offers rewards for meritorious performances and sits in judgment on the efforts of aspirants for distinction. It furnishes

the nearest approach we can expect to a fixed standard of excellence by which the work of new hands and the new work of old hands can be judged . . . There are a certain number of squinting brains, as there are of squinting eyes among every thousand of any population . . . We trust it will always be enough for a physician to be able to say 'I am a Member of the New York Academy of Medicine!'"

A few years later, on January 22, 1897, the semicentennial was celebrated. The list of the accomplishments of the Fellows of the Academy during its first fifty years is too long to enumerate in its entirety; however, they fought for the improvement in the preliminary education of matriculants, for State examination as a standard of license to practise, for increase in the duration and number of college courses and medical school inspection; they agitated for new factory laws in behalf of women and children, for clean streets, for improvement in tenement house conditions, school houses, Reception Hospital, reformation of quarantine for the Port of New York, water supply for the city (Croton water and watershed), establishment of the Metropolitan Health Board of the city, protection of the Port of New York and the city against invasion by cholera, and for the establishment of free baths. In fact, to quote directly from Dr. Jacobi's semicentennial oration, "The responsibility toward both the public and the profession was always deeply felt by the Academy. What I could say, fragmentary though it be, should have convinced you that the best individual and collective efforts of the profession, as represented in the Academy, are being spent in the *service of the community*. [The italics are mine]

"See to it that no personal interest, vanity, or misapprehension interfere with the progress of medicine. It is through our own efforts that we overcame the lack of knowledge on the part of legislators, and the opposition of medical schools, when we enforced a certain amount of preliminary education and the establishment of State examinations. See that these, your gains, for they are yours, be not taken away from you; they were conquered in your behalf."

Lots on 43 and 44 Streets, directly adjacent to the Academy property, were purchased in 1910, because of the steady and rapid growth of the Academy's activities and Library.

The majority of us are familiar with the recent history of the Academy. To the Standing Committees on Admission and Library have been added the Committees on Public Health Relations, Medical Education,

Sections, and Fellowship, and just tonight, Medical Information. With new problems, new responsibilities have developed, which, up to the present, we have met. We moved into this building in 1926, and we have since had to build an addition for the rare book room and much needed office space. Our Library, now over two hundred twenty-four thousand books and periodicals, is growing rapidly, and we shall soon need considerable additional stack room. We are now the fourth medical library in the world, in point of size, and second only to the Army Medical Library in Washington on this hemisphere.

I have attempted to give you a brief outline of our history, as we approach our hundredth anniversary. It is a story of continued progress. There are, among our Fellows, those who think we have expanded too much and should curtail our activities. We must admit that these are troublous times, but, for my part, I am strongly in favor of continuing to be true to the traditions and ideals which have become our established principles.

During my term of office, I should like to see a quickening interest in Academy affairs evinced by a much larger proportion of Fellows and Members, especially of the younger group, for excellence in any department can be procured only by the labors of a lifetime.

It is my hope that we may stir the consciousness of the public to the significance of the Academy as a public health counsellor. We must continue our growth; Foundations have helped us appreciably because of the scope of our activities in behalf of the physician and of the public; we have an obligation to those Foundations, to keep faith with them, by carrying on in the same direction; and we have an obligation to the people, to keep faith with them, by carrying on, as they have learned to rely on us to do.

The obligations we have accepted and discharged carry with them reciprocal responsibilities. We are, after all, in the same position as other institutions of learning, both special and general; such institutions are constantly appealing for help, to maintain the forward progress of their work. We want to be in position to do better work, to meet increasing demands upon us. If we do not continue to progress, we shall not remain static; we must inevitably give up many of the things which are so important to the community at large. What we have already accomplished, we have just reason to be proud of; but from whence we came and where we are is not nearly so important at this juncture as whither we are going.

HEAT LOSS FROM THE HUMAN BODY*

Harvey Lecture, December 15, 1938

EUGENE F. DU BOIS

THE Harvey Society was founded thirty-three years ago by a small group of scientists who were interested in stimulating the development of medicine, physiology, and allied subjects. One of the leaders, the man perhaps most closely identified with the Society, was Graham Lusk. To his desk in the old building of the Cornell Medical College came investigators from all parts of this country seeking advice and inspiration regarding their problems. He was interested in many phases of metabolism, but the controversy he loved most intensely was that which centered about the relationship of the basal metabolism to the surface area. It was this that inspired our studies on radiation, convection, and vaporization and the general campaign was planned by Graham Lusk shortly before his death in 1932 which checked but certainly did not end the influence of this great leader and rare spirit.

Graham Lusk paid a great deal of attention to heat loss as well as heat production. Between 1910 and 1933 most students of the respiratory metabolism concentrated on heat production. Since 1933 several groups in this country and Europe have made intensive studies on heat loss. There is a dispute as to which is the better viewpoint, reflecting in a rather amusing manner the constant struggle that is taking place in the body between heat loss and heat production. At certain times of the day heat production is predominant and heat loss does its best to catch up. At other times of the day heat loss is the controlling factor. This can be illustrated by a diagram which shows the curve for a twenty-four hour period of heat production and heat loss in one of the men who works in our laboratory (Fig. 1). These curves were constructed partly by temperature measurements and estimations as to activity. They are, therefore, semi-diagramatic. The solid line gives the approximate heat production, the dotted line the heat loss.

* From the Russell Sage Institute of Pathology in affiliation with the New York Hospital and Department of Medicine, Cornell University Medical College

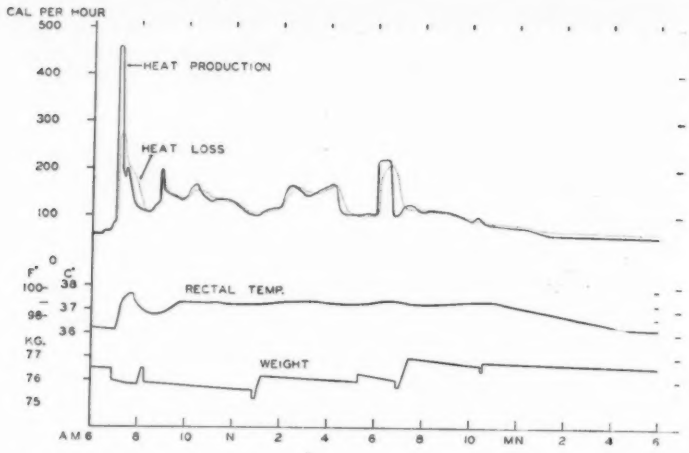

Fig. 1—An estimation of the variations which occur in the daily life of control D, obtained partly by direct measurements on several days and partly by estimation from calorimeter experiments on other days. The subject runs in the Park at 7:00 a.m. daily. The other peaks in the curve of heat production occur when he travels from his home to the laboratory in the morning, engages in the usual hospital activities, and walks home in the evening. The variations in weight are caused by meals, voiding, insensible perspiration, etc.

The day selected for the experiment was in the winter time and started at six o'clock in the morning with the man asleep in bed in a cold room. The rectal temperature was low. Heat loss and heat production balanced. At seven o'clock he got up, dressed, and ran one and one-quarter miles before breakfast. His basal metabolism was increased approximately six-fold; the heat loss was increased only four- or five-fold so that a considerable amount of heat was stored in the body with a resultant rise in the rectal temperature. While he was dressing and taking breakfast, the heat loss exceeded the heat production and the rectal temperature fell. The curve for the rest of the day showed the variations resulting from the walk to the laboratory, work, lunch, and the walk home followed by dinner, modified by the man's reactions in changing his clothing, opening windows, and other processes. During the night when he was asleep in a cold room, heat loss seemed to assume the dominant role. He did little or nothing to control the situation, and if his basal metabolism had not been set at a certain level he would have been as cold as a corpse before the night was over.

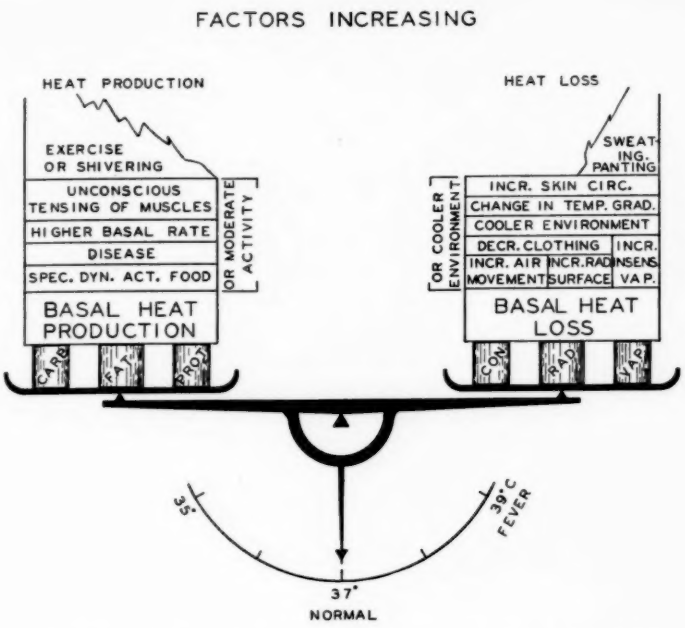

Fig. 2—Balance between factors increasing heat production and heat loss.

Another diagram (Fig. 2) which summarizes the whole situation is introduced at this point to bring out the multiplicity of factors. Heat production is placed on one pan of the balance, heat loss on the opposite pan. When the man is quiet in a warm room the basal heat production and loss equal each other and the body temperature is at its normal point of 37° C. When any other factor is added, the balance is temporarily altered until an equal addition on the other pan restores equilibrium. Heat production takes place only through the agencies of the combustion of carbohydrate, fat, and protein; heat loss through radiation, convection, and vaporization. When any one of these three factors changes on one side of the balance there is a readjustment of the other two factors. The details of these adjustments form the basis of this discussion. The major part of the problem is purely physical, but the conditions are frequently altered by physiological changes; and it is impossible and perhaps unimportant to make any sharp distinction between physics and physiology. It is well, however, to consider first

the body as a physical mass with an average internal temperature of 37° C, slightly warmer than this in certain organs. Since the body contains considerable amounts of fat and bone with low capacities, its specific heat is about 0.82, and a man weighing 70 kg. is thermally equivalent to 57 kg. of distilled water. The surface of the man is encased in a layer of skin and subcutaneous tissue which is unusually efficient as an insulator against heat or cold. The specific conductivity of cork is 0.0007 gm.cal./sec./cm.^{2}/cm.; epidermis, subcutaneous fat, and muscle tissue are slightly better insulators with conductivities between 0.00047 and 0.0005. The actual surface, as I shall explain later, is almost a perfect radiator or "black body" in the language of the physicist. On account of the configuration of the human body the surface area is large, for a 70 kg. man, about 1.8 square meters. Within 1 cm. of the surface of such a man there is a mass of tissue which weighs about 15 kg., about one-fifth of the total weight. When a naked man is exposed to an atmosphere of 22° C (72° F) there is a constant loss of heat through the surface which assumes an average temperature of about 30° C. This means that there is a gradient from the interior of the body to the surface, on an average 1.8 – 2.5 cm. deep, but it may in some places be as great as 10 cm. Inasmuch as the extremities have relatively small diameters and are situated at a considerable distance from the central heating apparatus of the body, their temperatures under cool conditions are considerably below the temperature of the central mass. In addition, the surface of the body is constantly moist, even when there is no activity of the sweat glands. Water from the moist interior seeps through the epidermis.

Of course man is more than such an inert mass of physical components. There is a constant production of heat in his muscles and organs. This heat is transferred to all parts of the body by means of the blood stream, warm blood coming from the muscles and the organs, cooler blood from the surface, producing relatively colder streaks as it passes through the veins of the extremities. In addition, there is a beautiful system of control of the blood flow to the surface so that the body can, when it is too cold, change the skin into a suit of clothes just as efficient as wool or cork. When the need for increased dissipation of heat arises, the body can pour quantities of blood into the deeper layers of the skin so that the inner side of the epidermis attains a temperature equal to that of the warmest portions of the body. At the same time

when the sweat glands pour out water, it can keep the surface moist enough to permit huge losses. Every gram of water vaporized from the surface of the body withdraws 0.58 calories through the latent heat of vaporization. Also, the body can change its heat production from a basal level of say 60 calories per hour to a level of more than 800 calories per hour by means of violent muscular exercise.

The skin is an organ beautifully adapted for its purpose. The tough epidermis is perforated by sweat glands, and beneath the Malpighian layer there is a rich plexus of capillaries, arterioles, and venules so that there can be a tremendous increase in the blood flow. Moreover, the skin is provided with sense organs for heat and cold, having a delicacy which was not suspected until Hardy and Oppel[1] proved that the skin of the forehead could detect changes of 0.003° C in three seconds when exposed to heat, with slightly lower sensitivity when exposed to cold.

MODE OF HEAT LOSS	TEMPERATURE OF SURFACE OF BODY T_s	TEMPERATURE OF SURROUNDING OBJECTS OR AIR T_a	AREA OF SURFACE OF BODY A	CHARACTER OF SURFACE E	SPECIAL FACTORS
RADIATION	$(T_s)^4$	$(T_a)^4$	85% EFFECTIVE	99% BLACK	
CONVECTION	T_s	T_a	100%	SMOOTHNESS	AIR MOVEMENT
VAPORIZATION	PERSPIRATION VAPOR PRESSURE T_s	HUMIDITY VAPOR PRESSURE T_a	100% FOR SEEPAGE + RESPIRATORY PASSAGES	% WETTED BY SWEAT + RESPIRATORY ACTIVITY	AIR MOVEMENT

Fig. 3—Simplified expression of laws of heat loss.

The importance of the surface area of the body is clearly brought out in Fig. 3 which is a simplification of the rather complex physical formulas for heat loss by radiation, convection, and vaporization. Newton's Law of heat loss considers only radiation and convection. Radiation is proportional to the temperature of the surface of the object minus the temperature of the surrounding objects or air multiplied by the surface area, corrected for the character or physical blackness of the surface. In the case of convection, air movement is of prime importance but this does not affect radiation. For radiation, the Stefan-Boltz-

man Law is more accurate, involving the fourth powers of the absolute temperatures of the surface and of the surrounding objects. These figures are obtained by finding the levels above the absolute zero, −273° C. For small differences such as we are discussing, the simple Newton Law is sufficiently accurate.

Vaporization is more complex depending on the difference between surface temperature and air temperature, or rather on the difference between the vapor pressure of the air immediately in contact with the slightly moist skin and the vapor pressure of water in the surrounding air, which is the expression of its humidity. Air movement is an important factor. Under ordinary conditions, about two-thirds of the water vaporized comes from the whole surface of the skin, one-third from the respiratory passages. The depth and frequency of respirations may become factors though they are not especially important.

It will be noted that each one of these modes of heat loss is proportional in some way to the total surface area of the body and this applies with modifications to all warm-blooded animals, birds as well as mammals. I have not time to go into the question of the surface area law but I shall refer you to Benedict's book on *Vital Energetics*[2] which compares calories per square meter of body surface for a large number of warm-blooded animals. Most of these show approximately the same level of basal metabolism per unit of surface area. A few are quite far off the line. I do not know where or when the various species of animals were given their basal metabolism. Perhaps Noah did it when they left the Ark. I suppose that the reason he could not do a uniform job with the animals was because he did not have scales small enough for the dwarf mice and large enough for the bull, the horse, and the elephant. The subject deserves more study.

There are a few minor heat losses which should be mentioned. The warming of food and drink is relatively unimportant. Conduction (*Leitung* of the Germans) or the transference of heat through solid objects is a factor if a man lies on the cold ground. Locally it becomes manifest when one touches a metal object and heat is transferred so rapidly from the skin that the object feels much colder than its non-metallic surroundings which are at the same temperature. In most of the older physiological literature the term conduction apparently includes heat loss by convection. I searched many of the textbooks of physiology before I found any mention of convection.

In our experimental procedures we have reduced conduction until it is practically zero. Heat lost in warming the air from the lungs has been added to the convection from the surface of the body and the vaporization from the lungs measured together with the vaporization from the skin.

When we made our first calorimeter experiments we were under the impression that radiation was the most important channel of heat loss. Later we discover that it is important chiefly under basal or quiet conditions. Still it is a constant factor deserving of much more attention than it has received.

The study of heat loss is probably as old as the hills. In a previous highly scientific publication I was able to prove that the main facts were discovered in the Garden of Eden by Adam and Eve within twenty-four hours of their creation. They probably learned a good deal more when they took to clothing and manual labor. Civilized man has developed a good practical knowledge of the subject, not quite as good as that of the Eskimos who are still in the early Stone Age. I suppose Newton was the man who did most on the subject of heat loss. It was Rubner who first grasped the real significance of the subject as far as man was concerned. He published in 1896[3] a table giving the measured or estimated radiation from a man producing 2700 calories a day, together with the estimated losses from convection, radiation, and the warming of food, and this is about the only table that has been published in the textbooks. His methods are not stated, but he grasped the essential points and made allowance for the fact that radiation does not depend on the total surface of the body but rather on its profile surface according to Lambert's Law. There is, for example, no loss of heat by radiation in the axilla or between the legs. Rubner's table of the partition of heat loss applied only to the special case of a man who was active enough to produce 2700 calories a day under special atmospheric conditions. His data were probably faulty, but Rubner was a genius who could draw correct conclusions from inaccurate and incomplete data whereas most of us draw incorrect or incomplete conclusions from data that are accurate.

Lefèvre[4] in France was a pioneer in this field of heat loss. In more recent years Cobet and Bramigk[5] and Bedford and Warner[6] have made good use of radiometers. The work that we found of most help was contained in the neglected publications of Aldrich[7] of the Smithsonian

Institute. He studied the surface temperature, radiation, and convection of school children but his instrument was rather bulky and slow. The long series of studies by Bohnenkamp[8] and his associates in Grafe's clinic in Würzburg have been most stimulating. Bohnenkamp was really the first man to emphasize the importance of the profile or effective surface area in relationship to radiation and this effective surface which amounts to 80 to 85 per cent of the total surface has been appropriately called the "Bohnenkamp surface." He brought out a good deal of evidence to show that heat loss as determined by effective surface area is a factor in setting the level for heat production. His radiometer had several faults of construction and many critics have pointed out errors in his work, focussing their attention on these errors rather than on the stimulating originality and significance of his main conclusions.

Since we have been engaged in our own studies several important publications have appeared. Burton[9] working with a calorimeter in Murlin's laboratory in Rochester has used a jacket made of resistance wires to measure the changes in skin temperature and has drawn valuable conclusions regarding the temperature gradients and the surface of the body. Winslow, Herrington and Gagge[10], using the splendid equipment of the Pierce Laboratory in New Haven, have been able to study the heat losses under a large variety of environmental conditions, securing wide differences between the relative proportions of radiation and convection. Their work has been characterized by a careful analysis of all the physical factors. They have brought out the conception of operative temperature and degree of wetness of the body, and they have developed the study of the conductance of the skin. Their papers will serve as a basis for the scientific study of air conditioning.

Time does not permit more than a superficial review of the literature with all of its ramifications. For example, it is impossible to cover the studies of Benedict and Carpenter of the Nutrition Laboratory in Boston who have been publishing on the subject of direct calorimetry since the days of the first Atwater-Rosa-Benedict calorimeter and have made valuable contributions on the subjects of skin temperature and heat loss by vaporization. The work of Bazett, McGlone and Burton[11] on the temperature of the muscles and subcutaneous tissues and temperature gradients is of fundamental character. The heating and ventilating engineers, especially Houghton, Sayers, Yant and Yaglou of the U. S. Bureau of Mines and Barker of England, have made studies of the

subject from a theoretical as well as a practical standpoint. Levine and Gordon and their associates[12], working first at the Nursery and Child's Hospital and later at the New York Hospital, by means of exceedingly careful and detailed studies, are furnishing the data for heat loss of small children, a field of clinical as well as theoretical importance. Hick, Keeton, and Glickman[13] in Chicago are making many studies of various physiological manifestations at different temperatures. Deighton[14] in 1933 wrote an excellent review of physical factors in body temperature and heat elimination. Perhaps the best work on the subject, a work too much neglected, is that of Leonard Hill[15] published in 1916-1919. Many of the points that he brought out in these publications are being rediscovered at frequent intervals.

Graham Lusk and his associates in the Department of Physiology of Cornell University Medical College have contributed a long series of papers on animal calorimetry starting in 1912. The Russell Sage Institute of Pathology, of which Graham Lusk was the Scientific Director, had been working since 1913 with an Atwater-Rosa-Benedict respiration calorimeter which measured heat production by the methods of indirect and direct calorimetry. The indirect or chemical method gave the total heat production with the details regarding the proportions of protein, fat, and carbohydrate oxidized every hour. The direct method gave independently the calories lost in vaporization and lumped together the calories of radiation plus convection. The sum of these, corrected for calories stored in or lost from the body, indicated the total heat production, using physical rather than chemical measurements. In the long run the two methods checked satisfactorily and served as an indication of mutual accuracy. The calorimeter was employed for nineteen years, chiefly in a study of heat production, but about 1931 we developed a great curiosity regarding the mechanism of heat loss, having published some data which were difficult to explain. For example, Barr and Du Bois[16] in 1918 in the study of malaria had stated that it was possible for the skin to give off more heat when it was cold than when it was warm. This seemed to be a violation of Newton's Law of cooling, and it brought forth a number of stinging comments which could not be dodged. After all we knew very little about the partition of heat loss from the human body. Heat, perhaps the most important end product of metabolism, had been neglected because there was no satisfactory method of dividing radiation from convection.

For many years we tried in vain to find a method for the direct measurement of convection, the heat carried away from the body in the moving currents of air. Physicists have always encountered great difficulties in this measurement and even the fundamental laws of convection are still vague. The factor of radiation, however, lent itself to exact measurement and its laws for inanimate objects were on a firm foundation. We therefore decided to work out a method for measuring the radiation from human subjects in the calorimeter and then determine convection by subtraction. Many radiometers had been devised but none was adapted to our needs. Fortunately we were able to find as a collaborator Dr. James D. Hardy, a physicist, who had worked in this field and possessed not only the technical skill but the insight which enabled him to perfect a radiometer and, what is more important, grasp its possibilities. It only required a year to produce a satisfactory model and another year to complete the tests which established its accuracy.

The Hardy radiometer[17] shown in Fig. 4 has been in use since 1934 but has been improved in several details until it is extraordinarily rapid and accurate. The radiation from a uniform area of skin is concentrated by means of a small silver cone on four or eight blackened thermojunctions made of extremely thin tin foil. A corresponding number of cold junctions lie just outside the range of radiation and compensate for changes in air temperature and air movement. Readings are made by means of a null-point galvanometer and potentiometer, and a scale has been constructed which gives both radiation and surface temperature. The apparatus is calibrated in a few minutes by means of a Leslie cube into the side of which has been inserted a blackened cone which is a perfect black body. Calibrations are made at the same temperature as that of the skin.

The measurement of radiation from the human body could be accomplished only after a prolonged campaign of investigation carried on by Dr. Hardy and his associates. First, it was necessary to establish the wave lengths of the emanations from the skin and to accomplish this Dr. Hardy designed an original and accurate spectrometer for the infrared which was constructed by our associate, Mr. G. F. Soderstrom, who seems to be able to make any instrument under the sun. This particular spectrometer happened to be beyond the sun because it was planned for the region far outside the influence of the sun's rays. A diagram, Fig. 5, based on Planck's law, gives in condensed and distorted form the whole

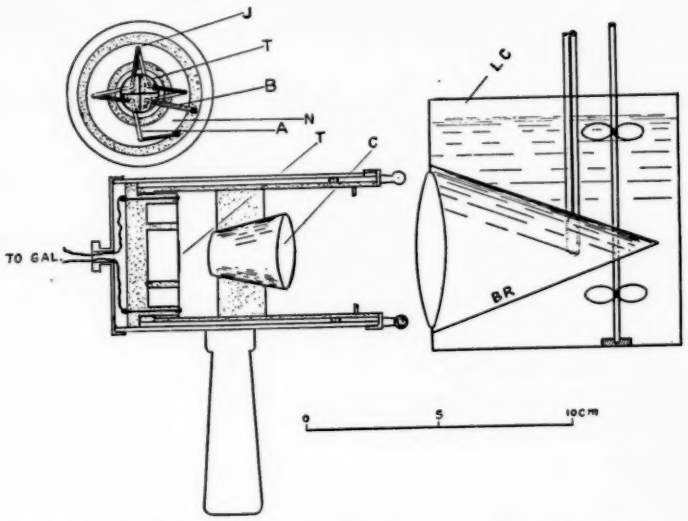

Fig. 4—Hardy Radiometer. Vertical section (lower left) and Leslie Cube (right). Detail of thermopile construction (upper left). T, blackened tinfoil which receives radiation concentrated by silver cone, C. J, cold thermojunction outside the zone of radiation. BR, copper cone laid into side of Leslie Cube (LC). The inside of this cone is painted a dull black and it constitutes a perfect black body radiator, the temperature of which is measured by means of the thermometer submerged in the water which fills the Leslie Cube.

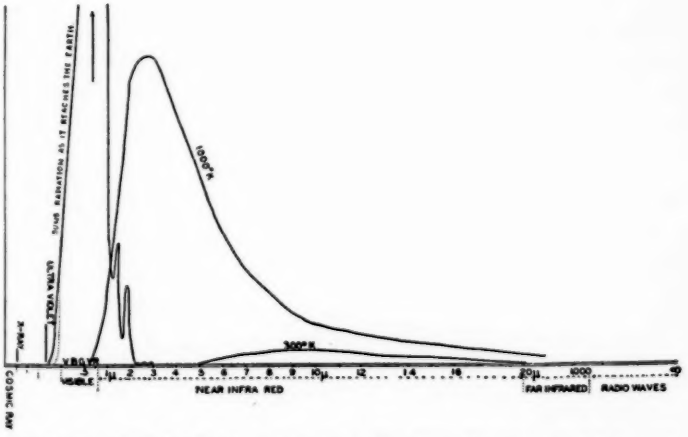

Fig. 5—Spectral distribution of the radiation from the sun, a red hot stove, and the human body.

range of wave lengths from the very short cosmic rays and x-rays on the left to the long infra-red and radio waves on the right. The radiation from the sun as shown in the highest peak starts in the ultraviolet, reaches its maximum in the visible range, and is shut off by the earth's atmosphere a short distance below the red at a wave length of about 2 to 3 μ. The radiation from a red hot stove begins in the red and extends into the far infra-red. The human body has a much lower temperature and therefore its emissions have wave lengths which fall between 5 and 20μ with a summit of about 9μ. These waves are stopped by many substances, such as glass, through which the sun's rays penetrate with ease. They fly outward with the speed of light in straight lines.

From the standpoint of radiation a "perfect black body" is a substance which reflects no rays, transmits no rays, but absorbs them all; or, if the heat be travelling in the opposite direction, radiates them all. In the visible range such objects necessarily look black to the eye but when it comes to the infra-red, color does not count. Hardy and Muschenheim[18] in this range found no essential difference between the skins of white men and coal black negroes. In a dark room in the middle of the night we are all black.

They found that human epidermis, even in layers that are extremely thin, blocks off almost completely the passage of infra-red rays longer than 3μ. The human skin comes within 1 or 2 per cent of being a perfect infra-red black body radiator. This confirms the findings of the majority of previous investigators and refutes some of the more recent work. The blackness of the skin is of considerable physiologic importance and it is particularly fortunate for the investigator since it enables him to calculate skin temperature from radiation.

The surface involved in the measurement of radiation is the profile or "Bohnenkamp surface" which in the case of a man in military or mummy position is very close to 80 per cent of the total surface as measured by the Sage Height-Weight formula. When a man squats with his knees close to his chest he can reduce his effective surface until it is only 55 per cent of the total surface.

One of the most important factors is the surface temperature and the literature on this subject is enormous. Many years ago G. N. Stewart pointed out that it was easy to measure the skin temperature but that it was hard to know what to do with it. Dr. Hardy has said and proved that it is very difficult to measure skin temperature but easy to know

what to do when you have it. In a series of careful experiments he demonstrated that the commonly used surface thermometers which are partly in contact with the skin and partly in contact with air or solid substances are subject to many errors. His method of using a radiometer to find the skin temperature without touching the skin is now generally accepted as the only method that gives absolute readings, although the other instruments may indicate differences with sufficient accuracy for clinical purposes. Dr. Hardy's applications of his exact measurement of skin temperature are so numerous that they would fill many chapters. Suffice it to say that they form the foundation of this report.

Just as important as the temperature and radiating properties of the skin are the temperature and character of the objects towards which the body radiates. In most rooms the air and walls are at the same temperature and the walls are fairly good black bodies. In many rooms there are spots of heat on radiators and spots of cold at the windows. Winslow, Herrington and Gagge[10] have made use of a combination of cool air and cool polished walls which reflect radiation from an electric stove. The experimental subject sits in a comfortable chair surrounded by polished copper walls, and the stoves reflect, from the polished copper, radiant heat without materially raising the temperature of the air. The New Haven investigators have been able to make a series of studies with the different radiating temperatures and have ingeniously calculated the "operative temperature" under many conditions.

Convection depends chiefly upon the rate and character of the movement of the air over the surface of the body. The flow may be uniform or turbulent, steady or intermittent. Losses by convection increase rapidly with the rate of movement of the air, but Leonard Hill[15] has shown that after a certain point the curve flattens. With speeds greater than 30 meters a second, approximately 70 miles an hour, there is no greater loss of heat through convection because the particles of air move so fast that they do not have time to take up more heat. Hill points out that this may be some consolation for motorists. In spite of the general belief to the contrary, the humidity of air has practically no effect on the loss by convection and metal objects lose heat at the same rate whether the air be humid or dry. With animals humidity would affect convection secondarily because it modifies vaporization and dampness probably makes the clothing or fur a better conductor of heat.

The study of perspiration is fascinating and Kuno[19] of Japan has written a book in English on this subject. Inhabitants of northern climates possess two or three million sweat glands, those who dwell in the tropics about a million more. On the hands, feet, and axillae there is a continual slight activity of the glands. Over the rest of the body the eccrine sweat glands are called into action when needed to dissipate heat. There are some areas, such as the palms of the hands and soles of the feet, which respond promptly to emotion but lag behind when the rest of the body is trying to lose calories. The apocrine sweat glands in the axillae and pubic regions are a good deal of a nuisance because they secrete those specific aromatic substances which contribute more than their share of body odor. The eccrine glands over the rest of the body are man's safety valves in hot weather and there is no one human attribute of more importance than the ability to sweat skilfully.

In cold weather the humidity of dwellings is always low and there is such a large gradient between the vapor pressure of the skin and air that vaporization is rapid. Even a large rise in the percentage humidity of a room at 20° C (68° F) makes very little difference in vaporization, and after all, vaporization at this temperature accounts for only one-quarter of the total heat loss.

I have reviewed the fundamental laws of heat loss in the light of our present understanding but I must confess that when we started work there was a great confusion in the literature and in our own minds. It was only as we progressed from experiment to experiment and compared our results with those of others that the subject became relatively simple. We have been forced to change our views many times and are fully prepared to change them every year. It was in 1932 that the Russell Sage Institute of Pathology moved its calorimeter to the New York Hospital where a room had been provided with adequate temperature control. In 1934 the radiometer had been fully tested and we were in a position to make the first studies in which heat production, total heat loss, radiation, convection, vaporization, surface temperature, and storage of heat in the body could be measured accurately.[20] We were confronted with many variables and decided to reduce them to a minimum. In order to keep heat production constant it was necessary to work with subjects under basal conditions. In order to measure surface area and skin temperature we decided to use naked subjects. In order to eliminate conduction we employed a bed made of fish line, using a

small folded sheet under the hips and shoulders and a small towel under the head. Since the men were lying close to the bottom of the calorimeter and the currents of air were directed across the top, convection was reduced to the lowest possible level. The velocity of air near the surface of the body was too small to be measured by delicate anemometers or appreciated by the skin. There remained, however, a natural convection current due to the warming of the air by the man's body. It is necessary to emphasize these points because other observers have used patients after food intake and in different positions. Any such departure from the basal, causes wide differences in the results.

Our main studies were made on two normal men and three normal women, supplemented by a small number of experiments on other normal controls including nudists.

The routine for the day was as follows: The subject of the experiment came to the laboratory about nine o'clock, having eaten no food since the previous evening. He sat in the calorimeter room at the experimental temperature for about an hour, and then as he undressed, surface temperature readings were made at twenty different areas on the skin. He was then weighed and his height measured so that we could calculate surface area. Next, he was placed on the calorimeter bed, the electrical thermometer inserted 12 cm. into the rectum, and the radiometer hung on the inside of the calorimeter. About eleven o'clock the box was sealed and the preliminary period started. About twelve minutes before twelve o'clock, after an equilibrium was established, the man was instructed to start the second series of the skin temperature readings. This he accomplished by pointing the radiometer at the same twenty spots of the surface. These measurements required about two minutes and involved the expenditure of 1 to 2 calories. Next followed a quiet period of ten minutes, and the first experimental period was started at about noon. During the first hour the man lay motionless. At one o'clock this basal period was terminated and a transition period of twenty to thirty-five minutes started, at the beginning and end of which we measured surface temperature once more. This was sometimes followed by a second basal hour, but in a good many experiments a new factor was started in the transition period such as moderate work or the use of the electric fan. In the colder experiments the procedure was modified so that the transition period accommodated itself to the spontaneous chill. The subjects became very skilful in giving us a warning

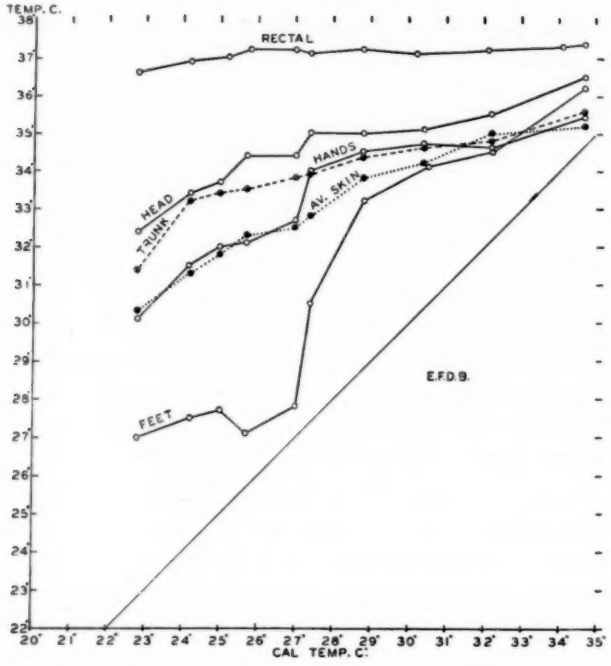

Fig. 6—Skin and rectal temperatures of subject D, during basal experiments at different environmental temperatures.

about ten minutes before the chill became inevitable. After the third period, terminating about 2:30 p.m., we measured surface temperature once more, and the man was taken out of the box and given a much needed breakfast.

The first problem was to determine the basal heat production and heat loss over the greatest possible temperature range. We could not use room and calorimeter temperatures higher than 36° C (97° F) because the sweating would be too great for us to measure accurately in our calorimeter. We could not go below 22° C (72° F) because it was impossible for the man to remain quiet long enough before having a chill. This is ordinary room temperature, and it does not seem cold to us who are clothed and moving about, but it does feel cold when you are lying motionless with an empty stomach and a naked body. Even trained artists' models and nudists shiver under these conditions.

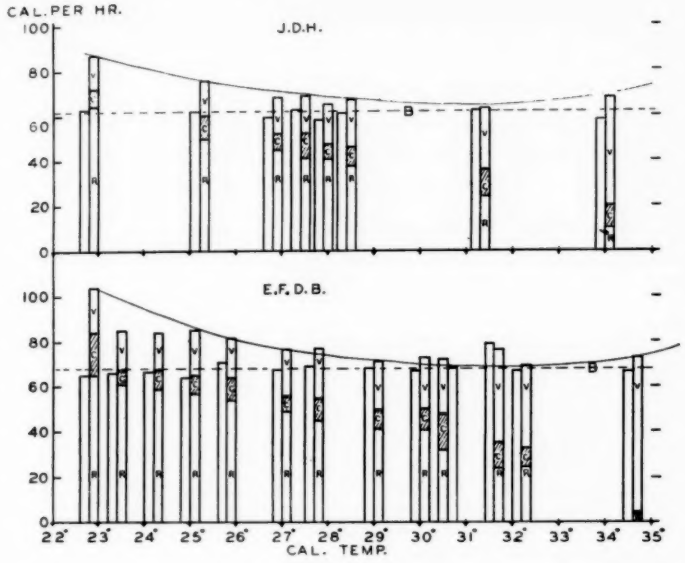

Fig. 7—Changes in heat production and heat elimination for two male subjects during the basal periods with increasing calorimeter temperatures. Blank columns on left, heat produced; columns on right, heat eliminated; V, vaporization; C, convection; R, radiation.

Having secured the basal metabolism in the first experimental period it was easy to add for comparison the other factors previously mentioned. The basal periods were fairly simple, but when it came to chills everyone was busy, almost as busy as the man who was having the chill. Personally I find it much easier to have a chill than measure one. When the experiment was over, it required a particularly long series of calculations to determine radiation. It was necessary to find the average surface temperature and effective surface area of each part of the body. Fig. 6 gives the average temperature for one of the men measured after he had been lying naked and motionless for 2 to 3 hours. The straight diagonal gives the air temperature, the top line the rectal temperature. In the warm zone of 34° C (93° F) all temperatures are within 2.5 degrees of each other. In the cold experiments the differences are great and the temperature can fall in large masses of body tissue without materially affecting the rectal temperature. The feet are almost

as cold as the surrounding air. In fact, the surface of the toes is usually 1 or 2 degrees below air temperature. Their constant moisture causes excessive cooling. They look and act like good radiators.

Fig. 7 gives the results on the two men who have been studied at frequent intervals in the calorimeter. The first blank column for each experiment represents heat production as measured by oxygen consumption. It will be noted that the basal metabolism was surprisingly level with no sign of Rubner's "chemical regulation." Even at the coldest temperatures it did not rise until about five minutes before a frank chill. The second columns show heat loss divided into radiation, convection, and vaporization. Inasmuch as radiation depended on the temperature difference between the surface of the skin and that of the walls, the heat loss by radiation diminished uniformly with rising temperature until it disappeared completely when skin and walls were at the same level.

Convection showed no marked diminution until 32° C. Like radiation it disappeared when the air was as warm as the skin.

Vaporization accounted for 18 to 20 per cent of the heat loss at the lower levels, 25 to 40 per cent in the middle zone, increasing rapidly above 31° C until it was obliged to take over 100 per cent of the heat loss at 35° C, since radiation and convection had fallen to zero.

The temperature range studied must be divided into three zones. The warm zone above 30° C is a region of discomfort because the skin is dripping sweat. The man is too hot and the balance between heat loss and heat production is made but poorly. There is a region between 30° C and 28° C where the heat loss is equal to the heat production. This is the so-called comfort zone, and it is comfortable because the body is able to make quite easily the physiological adjustments which maintain equilibrium. In the warm part of this zone the amount of blood flowing to the skin is so large that a great deal of heat is carried close to the epidermis. The sweat glands are not called into action. In the colder part of this comfort zone the blood flow to the surface is so greatly diminished that the skin has become as good an insulator as a suit of clothes.

Below 28° C is the zone where heat loss inevitably exceeds heat production if the man remain in the basal state. As the atmosphere grows colder, vaporization and convection remain about the same but radiation increases and there is nothing that the naked body can do to check this loss. At the lower limit of the comfort zone there had been a maximal

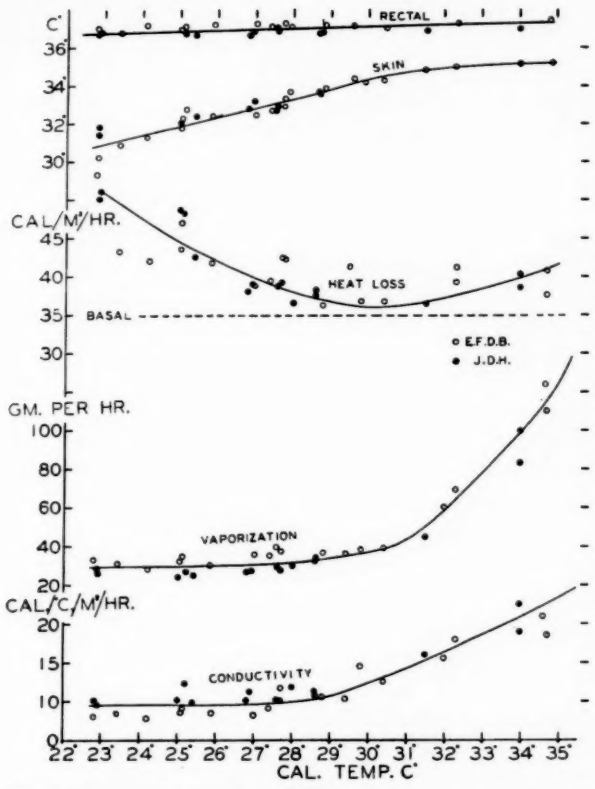

Fig. 8—Changes in factors involved in heat loss regulation with increasing calorimeter temperatures.

constriction of the subcutaneous vessels. The skin had become a suit of clothes but even a suit of clothes has its limitations. Below this level the living body loses heat like an inanimate object. Some people, especially trained nudists, boast of their ability to stand cold weather but nudists possess no magic that will interfere with the laws of radiation.

Fig. 8 brings this out in a more graphic form, giving once more the temperature range from 22 to 35° C. The top line shows the rectal temperature slightly lower in the cold zones than in the warmer. The skin temperature, however, is much lower in the colder zones with a curve which flattens out in air above 31° C. The heat loss is far above the basal heat production in the colder zone, the vaporization curve

makes a sharp break at the upper limit of the comfort zone about 31° C. The line for the conductance of the skin is the most interesting. Below 28° C it is practically straight and this means that throughout this region the skin has just the same conductivity because it is almost bloodless and acts like a dead insulator, slightly more efficient than cork. Above 28° C it is evident that a new factor has been added, transferring heat from the interior of the body to the surface and this new factor is, of course, an increasing amount of blood. The blood flow to the skin can be measured by this curve, and it has been found that with an environmental temperature of 34° C the cutaneous circulation may account for 12 per cent of the total cardiac output.

Last year in the Sage Laboratory, Dr. Hardy and Dr. Milhorat made a similar series of calorimeter observations on three healthy normal women of average build. They have kindly given me permission to discuss their results in advance of their publication. The rectal temperatures are the same as those of the two men, skin temperatures a little lower in the cold zone, higher in the warm zone. Basal heat production, much to the surprise of all of us was the same as that of the men in the colder ranges but dropped very markedly between 28° C and 31° C. Heat loss which was lower than that of the men in the cold zone showed exactly the dip in the warmer zone that had been evident in the basal metabolism. The conductance of the skin was lower than that of the men, as might be expected from the slightly greater amount of subcutaneous fat. The thermal loss or loss of heat per square centimeter of surface for each degree of difference between the skin and environment was absolutely uniform both for men and women throughout this range, demonstrating the fact that the experimental procedure possessed an accuracy that compared most favorably with that obtained by physicists working with inanimate objects.

This curious dip in basal metabolism in warm environments may be considered a type of chemical regulation, changing the metabolism without any change in the physical activity. There seems to be no question about it since the methods of direct and indirect calorimetry agree almost exactly. It may perhaps explain the reason that there has been so much uncertainty regarding the level of the average basal metabolism of women. After all, the differences between men and women shown in this chart are in accord with the general observation that women stand cold much better than men. Perhaps it also brings out the point known to the

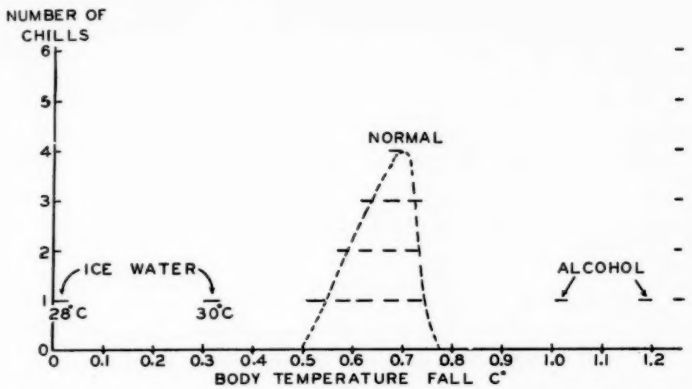

Fig. 9—Diagram showing the fall in the average body temperature before the onset of a frank chill. Under normal conditions the chill comes after a drop of about 0.65° C. Alcohol postpones the chill, ice water in the stomach precipitates the chill.

ancients: "*Varium et mutabile semper femina.*"

One of the most striking facts in these experiments is the great excess of heat loss over heat production in the colder atmospheres. Obviously, the naked person exposed to air at 22 to 24° C cannot go on losing heat indefinitely. The body reacts with a chill which is purely involuntary. Thirteen such "normal chills" have been observed in our experiments in men and women (Fig. 9). We calculate the average body temperature fall, giving a weight of 80 per cent to the mass of tissue measured by the rectal thermometer and the other 20 per cent to the superficial tissue represented by the average skin temperature. It is evident that these chills come with surprising regularity when the average body temperature has dropped two-thirds of one degree. This, of course, applies only to chill experiments with naked subjects under our experimental conditions. If a drink of 60 cc. of brandy containing about 28 cc. of alcohol be given, the skin temperature is raised a little, the man feels warmer, and the chills are delayed until a fall in body temperature is almost twice as great. This may at first seem like a beneficial effect, but it should be considered as a serious and perhaps dangerous impairment of the temperature regulating mechanism. If, instead of giving alcohol, we substitute its anemic rival, ice water, we obtain a chill long before the chill would be due in the natural course of events.

We have studied in detail nineteen chills and have compared them with the chills of malaria and the chills following the intravenous injection of foreign protein observed by us twenty years ago in the same calorimeter. A sample chill shown in Fig. 10 followed a basal hour at an environmental temperature of 23° C (73° F). When the man came to the laboratory and undressed, his average skin temperature was at the usual level of 33.5° C. This dropped steadily during the preliminary period and first basal hour, the rectal temperature scarcely fell at all. Between 11 a.m. and noon his basal metabolism was at its normal level, about 60 calories, but inasmuch as the surface of the body was losing heat, the total heat loss was 28 calories greater than the production. The man felt no serious discomfort and no tensing of the muscles until 11:50 a.m., when he signalled that he felt sure a chill was due in about ten minutes. This gave us time to start another period in the calorimeter, and in a few minutes he experienced a paroxysm which shook the whole apparatus. This lasted twenty-five minutes and during the total period of thirty-six minutes the heat production was at the rate of 140 calories per hour, exceeding the heat loss by 43 calories. Most of this heat was stored near the surface, and the skin temperature rose distinctly with only slight change in the rectal temperature. After the chill the man felt comfortable, his heat production dropped to the basal level, but heat loss still remained high, the skin temperature fell abruptly, and he had a second chill a few minutes after the experiment was terminated. It is interesting to study the details of the heat loss in these three periods. The total loss was increased slightly during the chill and all of the increase could be ascribed to a rise in convection caused by the clonic movements of the body. Radiation was decreased because the skin temperature was lower during the chill than either of the basal periods. This is a confirmation of our much disputed statement made twenty years ago that the body could give off more heat through a cold skin than through a warm skin. Until we had the radiometer it was impossible to measure convection and none of us had the intelligence to realize the importance of increased air movement which would inevitably follow shivering.

The effect of moderate work is shown in the second diagram of Fig. 10, a sample of a large number of experiments. This observation was performed in the comfort zone at 30° C. The rectal temperature was constant in the basal hour. Heat production shown by the dotted line was the same as heat loss. In the short transition period and in the subse-

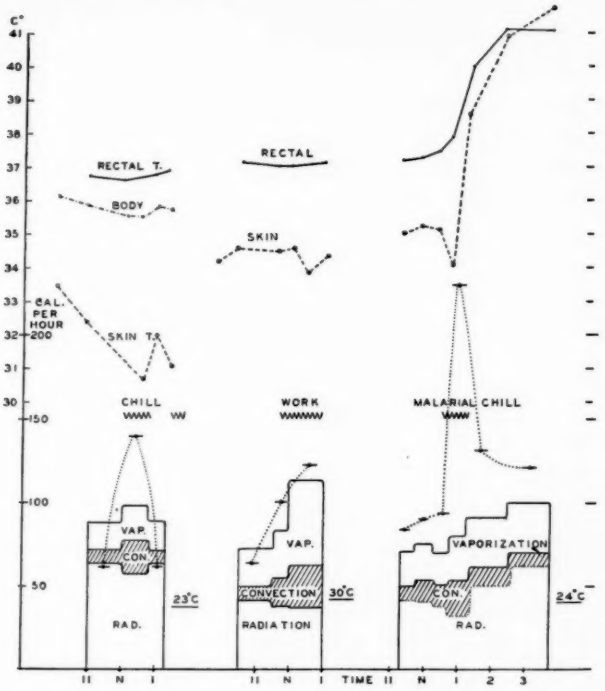

Fig. 10—Chills and Muscular Exercise. The first diagram shows chills which occurred spontaneously when the naked man was lying motionless in the calorimeter at a temperature of 23° C. The dashed lines connected by dots show heat production, the columns heat loss.

The second diagram shows the effect of moderate exercise at a temperature of 30° C.

The third diagram represents a malarial chill studied in 1918. There were no direct determinations of skin temperature or radiation but estimations have been supplied from later experiments.

quent longer period when moderate work was performed, heat production increased and heat loss kept pace so that the storage of heat in the body was scarcely affected. When work was started convection rose and in the third period there was a slight outbreak of sweat, increasing vaporization markedly. The rise in convection and vaporization cooled the skin so that radiation was decreased, proving once more that it was possible to lose more heat through a cool skin than through a warm skin. This experiment demonstrates nicely the delicate regulation of the tem-

perature but here it was an adjustment of heat loss through greater vaporization in order to balance the increasing heat production.

The third diagram of Fig. 10 is a recalculation of the often quoted case of George S. studied by Barr and Du Bois[16] in the calorimeter in 1918. At that time we did not have a radiometer and the dashed line indicating changes in the skin temperature and the blocks for radiation are reconstructed from evidence that we have obtained on other patients with chills. They are only approximate.

George S. came to Bellevue with a history of chills, recurring at regular intervals. We placed him in the calorimeter about two hours before an expected chill and measured his heat production and heat loss. In the first two periods his temperature was normal and heat production about 90 calories per hour, his heat loss a trifle lower. In the third period with the onset of a violent chill which lasted thirty-five minutes, heat production rose to 250 calories per hour, heat loss was almost unchanged, and he stored 113 calories in his body with an abrupt rise in rectal temperature to about 40° C (104° F). After the subsidence of the chill his metabolism fell but did not quite reach his previous level. He continued to store a little heat in the body. After the calorimeter observations he began to sweat, heat loss exceeded production, and the body temperature fell to normal. Judging from other chills, convection must have increased; surface temperature and radiation must have fallen. We have observed in other chills a rise in skin temperature until in some patients it is even higher than the rectal temperature.

Here the temperature regulation was very different from that observed in two other situations shown in this figure. In all probability the disruption of the malarial parasites had caused a sudden change in the level to which the temperature regulating center was adjusted. The center demanded a body temperature of 40-41° C and it found the body 4 degrees too cold. Therefore it summoned its emergency mechanism, a chill, and in the course of an hour or two accomplished the desired result. This, of course, was aided by the fact that the skin was kept relatively cold and heat loss maintained at or near a minimum. Following the chill the regulatory center desired a return to the normal level of temperature and accomplished this by calling upon its emergency mechanism of sweating, aided to a slight extent by increasing radiation from an unusually warm skin. Increased radiation, however, seldom adds more than 15 calories per hour.

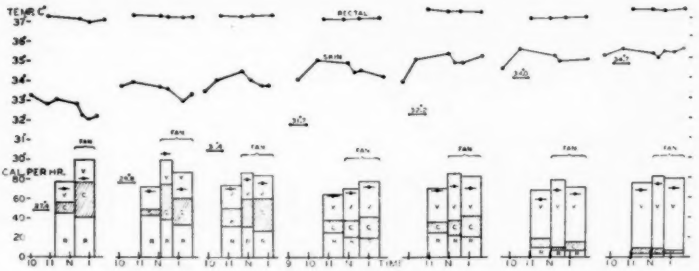

Fig. 11—Experiments with an electric fan. After one basal hour the large fan at the foot of the calorimeter was started. Convection was increased greatly except in the experiment when the air was as warm as the skin.

Passing from the chill experiments, the next factor to be considered is air movement. In a number of our experiments we started a large electric fan at the foot of the calorimeter shortly after the termination of the basal period. In Fig. 11 the observations are arranged according to ascending temperatures between 27.4° and 34.7° C. The first column in each case shows the heat loss in the basal period. It will be noted that in the colder experiments the fan caused a rise in heat loss above the level of heat production. This resulted in a fall in skin temperature due chiefly to the increased convection. In the middle zone there was almost always a slight fall in skin temperature caused by the fan. In the warmest experiment at 34.7° C when the air temperature and skin temperature were practically the same there was no radiation, convection was unchanged, vaporization accounted for almost the entire heat loss, and the total dissipation of calories from the body was not affected. Skin temperature and rectal temperature remained level. The subject's notes made during the experiment stated that he felt a little less uncomfortable when the fan was blowing. What did the fan accomplish? It did nothing except make a buzzing noise which aroused pleasant expectations in the mind of the subject.

The factor of exercise deserves more attention. In some experiments in the colder zone moderate exercise was introduced immediately after the first basal hour. Just as in the case of chills this muscular activity warmed up the body. The surplus heat was retained until the depleted heat reservoir had been filled and then the excess was dissipated by various channels.

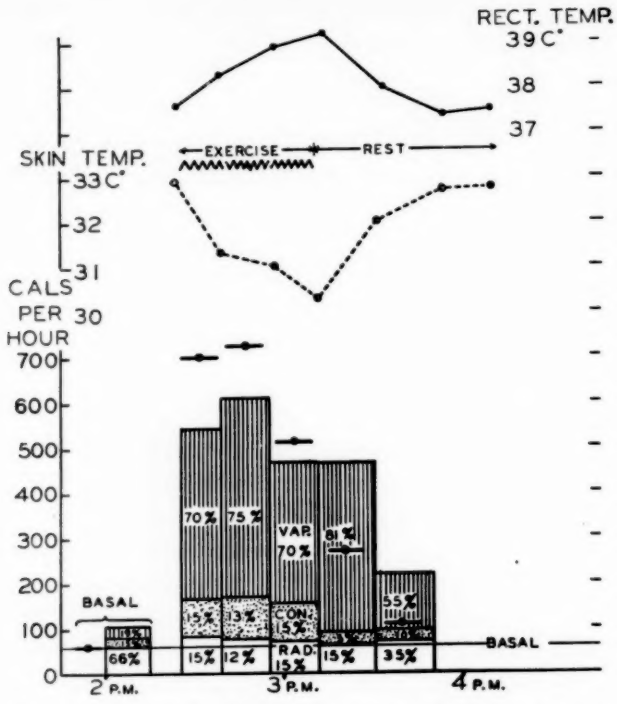

Fig. 12—Diagram showing the effect of violent exercise. Normal control JDH played three games of squash racquets. The rectal temperature rose sharply to fever levels, the skin temperature fell. The round dots with horizontal lines through them show the estimated heat production, and the columns the estimated heat loss.

Our calorimeter is not large enough to measure more than mild activity but we were able to obtain outside the calorimeter fairly good estimates of heat loss during violent exercise. Two of the staff played three twelve-minute games of squash-racquets in a court at a temperature of 20° C. Radiation, skin temperature, and rectal temperature were measured every twelve mintes. Vaporization was estimated by weighing the man every twelve minutes and making allowance for the small amount of sweat that dripped from the body. Heat production could be calculated roughly from these factors. The results on one man are shown in Fig. 12. The men were excellent players and they exerted themselves to the utmost. Heat production rose to the rate of 600-800 calories per hour but heat loss did not keep pace. The average skin temperature fell three

degrees and therefore radiation decreased. Convection rose markedly as the men rushed about the court. The great bulk of the burden fell on vaporization but even this was lagging under an excessive demand. The body did not lose heat fast enough and the rectal temperature rose three degrees.

As soon as the men stopped playing and rested in the dressing room heat loss was able to exceed heat production. Vaporization remained high, and in thirty-five minutes all the excess of heat had been dissipated. The skin temperature rose to its former level and the rectal temperature fell to normal. Soon after the game started the skins of the players had turned pink and it was evident that there was an increased flow of blood just beneath the surface. With a rectal temperature of 39° C and an average skin temperature of 31° C, there was a gradient of eight degrees between the moist surface of the body and the blood just beneath the epidermis. There must have been a tremendous interchange of heat in the zone one-half or 1 mm. deep.

Until a few days ago we held the opinion that the rise in rectal temperature during exercise was due to the inability of the body to lose heat fast enough. A very recent publication by Marius Nielsen[21] of Krogh's laboratory forces us to change our views on this subject. In a series of beautiful experiments with a bicycle ergometer he has shown that the rectal temperature rises rapidly when work begins and then tends to flatten out at a level determined by the severity of the exercise. Drastic changes in the temperature and movement of the external air make little or no difference in the level to which the body adjusts itself. The fact that when this level is attained during severe work the men can lose heat as rapidly as it is produced proves that during the milder grades the body had not employed its utmost capacity in dissipating calories. Nielsen has shown that during work the temperature regulating center is adjusted at a higher level and maintains the body very nicely at this level.

We have not time to survey our limited number of experiments on the effects of clothing. Suffice it to say that we have found that ordinary clothing does not change the fundamental concepts regarding the proportions of heat lost by the various channels but clothing does extend the comfort zone far into the colder ranges. The matter has been thoroughly studied recently by Winslow, Herrington and Gagge.[10]

This detailed investigation of heat loss through the various channels has rather important bearings on the problems of heating and air con-

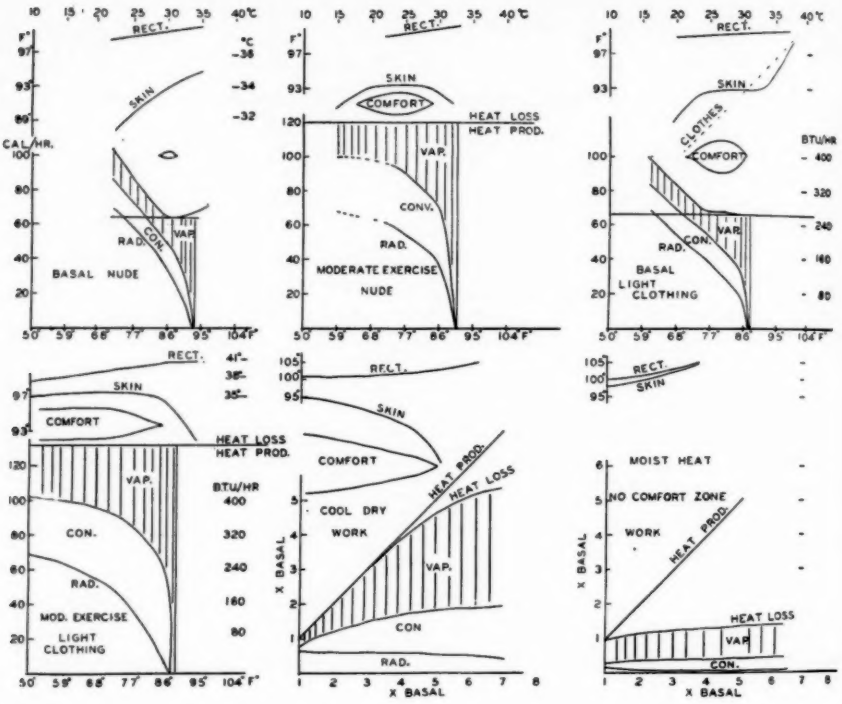

Fig. 13
COMFORT ZONES UNDER VARIOUS CONDITIONS

1. Basal nude. Narrow comfort zone where heat loss and heat production are equal.

2. Moderate exercise, nude. Comfort zone wider and at a lower range.

3. Basal with light clothing. Comfort zone at a lower range than in the case of the nude man. In the colder part of this zone the skin is warmer than the surface of the clothing; in the warmer part of the zone the skin is cooler than the clothing.

4. Moderate exercise with light clothing. Comfort zone extends far into the cold range.

5. Work in a cool, dry atmosphere. Abscissae show heat production in terms of the basal metabolism, six times basal representing moderately severe exercise. The ordinates show heat loss. The comfort zone is wide.

6. Work in a warm, very humid climate. Vaporization is impaired to such an extent that heat is retained in the body. There is no comfort zone.

[1 and 2 are based on an adequate number of calorimeter experiments; 3 and 4 on a few observations of our own; and 5 and 6 are taken from the work of other observers.]

ditioning of rooms. In order to outline some of these problems to serve as a basis of further studies we have sketched out various conditions in Fig. 13. The first two diagrams are based on a considerable number of

experiments, the second two with clothing on relatively few subjects, and the last two are largely theoretical, based on the work of other observers. The first diagram sketches the conditions shown in Fig. 9 based on the study of the two naked men in air temperatures from 70 to 94° F. There was a small comfort zone where heat loss equalled heat production. The second figure shows the conditions when the naked men were exercising in the calorimeter. Heat loss and heat production equalled each other over a wider range at a lower temperature, and the comfort zone was much more extensive. The third chart shows the conditions when the men were lightly dressed. The surface temperature of the clothes was roughly proportional to but distinctly above the level of the air while the skin temperature was warmer than the surface of the clothes in the cold zone, cooler in the hot zone when the man was sweating. The region of comfort as we all know was much more extensive than in the case of the naked man. The fourth diagram shows the combination of light clothing and moderate exercise with a comfort zone extending far into the cold region, depending on the extent of clothing and degree of exercise.

The last two diagrams on this chart are gathered from studies in the literature with extrapolations from our own work. In them the abscissae show the extent of work expressed in terms of the basal metabolism, six times basal representing fairly severe exercise. The ordinates show heat loss in terms of basal metabolism. When a man works in cool, dry air, the rectal temperature rises depending on the severity of the work, the skin becomes cooler, heat loss eventually becomes equal to heat production, the comfort zone is wide until the exercise is excessive. When, however, work is performed in a hot, moist atmosphere, the vaporization is no longer able to dissipate more than a small amount of heat from the surface of the body. Heat loss lags farther and farther behind heat production with increased amounts of work. Rectal and skin temperatures rise rapidly. The comfort zone is conspicuous by its absence.

Time does not permit the discussion of the temperature regulating center in the hypothalamus. It seems to be controlled by two factors, the temperature of the blood reaching the brain and the changes in the temperature of the skin. The pathways for the sensory connections from the skin through the spinal cord to the brain and then back from the brain to the skin, muscles, and internal organs are fairly well known. The internal mechanism of the small group of cells constituting the regulating center

remains a fascinating mystery.

This review of the subject of temperature regulation and heat loss is necessarily incomplete, but Giaja[22] has recently published an excellent survey. The field of study has been particularly active during the last few years and the advances have been great, chiefly on account of the fact that well trained biophysicists have been induced to participate in these studies. How far the modern investigations will take us is difficult to estimate. We shall undoubtedly learn more about the thermo-regulating mechanism; we shall learn more about the blood flow of the skin and its nervous control, and the clinical applications of this will probably be numerous; we shall get a better insight into the mechanism of fever and heat regulation during muscular exercise. We shall have a much better approach to the problem of comparing the basal metabolism of various species of animals. We shall learn more about artificial fevers and also obtain information that will help our friends, the air-conditioning engineers. It is doubtful if any of this work will have the slightest influence on clothing. In this field custom and fashion reign supreme.

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THE MEDICAL-SURGICAL SPLENOPATHIES*

Introduction

ALLEN O. WHIPPLE

SOME TEN years ago a group of internists, pathologists and surgeons agreed to pool their interests in diseases of the spleen and hematopoietic system and the Combined Spleen Clinic, as it is now called, was started at the Columbia Medical Center. The advantages of seeing patients with such disorders before, during and after the therapy had been agreed to, whether medical, surgical or roentgenological, soon became apparent, and may be summed up under three headings:

1. Advantages to the patients are definite in having a more active concentrated study of their lesions, a more accurate diagnosis, a therapy agreed upon by the group, and lastly a follow-up by the same group to determine the late results as a basis for advice to future patients.
2. Advantages to the members of the Clinic, as clinicians and teachers, are evident in the study of an increasing number of patients and in clarifying their ideas in diagnosis, pathology, therapy and prognosis.
3. The stimulus to clinical and experimental investigation of problems arising in the diagnosis and treatment of patients seen in the Clinic is obvious.

During the past ten years we have seen 1,457 patients with the lesions as classified in Table I. It is obvious that many of these blood and spleen dyscrasias are strictly medical in their therapy, others roentgenological, others surgical, and a fairly definite group where therapy is difficult to advise. The leukemias and primary anemias are studied and cared for on the medical services by Doctors McAlpin and West. The splenomegalies, of known and undetermined etiology, are studied by the combined group.

The organization of the Clinic is of importance and is graphically

* From the Department of Surgery, Columbia University College of Physicians and Surgeons and the Spleen Clinic of the Presbyterian Hospital, New York City
Delivered November 4, 1938 in the Eleventh Annual Graduate Fortnight

TABLE I

NUMBER OF CASES TO OCTOBER, 1938

Type	*Cases*		
Hemolytic Jaundice (typical)	43	30 with	splenectomy
		13 without	"
Hemolytic Jaundice (atypical)	15	7 with	"
		8 without	"
Normal Splenectomy	30		
Splenomegaly of Undetermined Origin	47	43 with	"
		4 without	"
Banti's Syndrome			
(1) Cirrhosis	64	22 with	"
		42 without	"
(2) Schistosomiasis	11	11 with	"
(3) Outside Pressure on Splenic Vein	3		
(4) Splenic Vein Thrombosis	8	3 with	"
		5 without	"
(5) Cavernomatous Transformation of Portal Vein	2	2 with	"
(6) Stenosis of Portal Vein	1	1 with	"
(7) Obstructive Factor Undetermined	33	22 with	"
		11 without	"
Idio-thrombocytopenic Purpura	42	22 with	"
		20 without	"
Miscellaneous Purpura	49	11 with	"
		38 without	"
Aplastic Anemia	54		
Pernicious Anemia	231		
Secondary Anemia	84		
Sickle Cell Anemia	10		
Miscellaneous Anemias	88		
Eosinophilia	13		
Hemophilia	15		
Infectious Mononucleosis	71		
Acute Leukemia	79		
Chronic Myeloid Leukemia	98		
Chronic Lymphatic Leukemia	89		
Hodgkin's Disease	141		
Neoplasm	70		
Polycythemia	66		

presented in Chart I. It is to be emphasized that the general practitioner and family physician is the beginning and end of this scheme. It is he who, as a rule, is first consulted and sends the patient for opinion and consultation and it is to him that we try to return the patient and whose co-operation we seek in having the patient return for follow-up studies.

The second feature of this organization that we wish to bring to your attention is the central and essential laboratory for the hematological studies of these patients. Before, during and after the therapy used in the individual case, the blood counts and various blood studies should be done by the same group of carefully trained and experienced hematologists. Unless this is done, the records in the Clinic and the conclusions drawn from the study of the patients will be thoroughly unreliable.

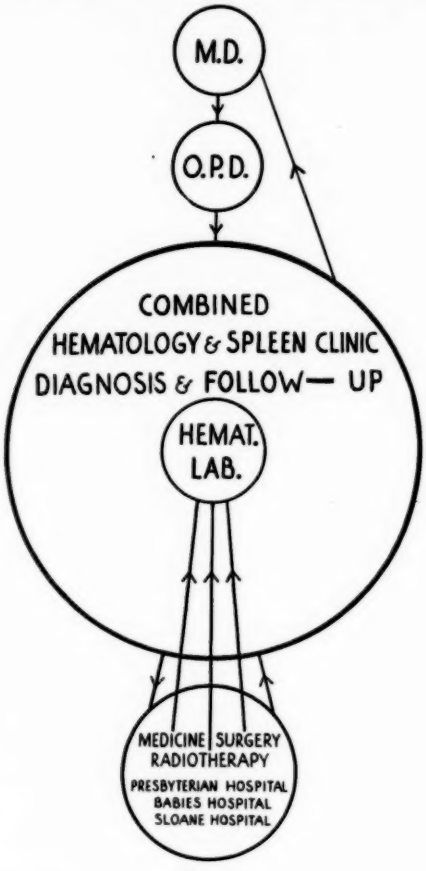

Chart 1

It is the desire of the members of this Clinic to pay tribute to the accurate work and interested co-operation of Miss Katherine Edsall, Miss Mary Whipple, Miss Catherine Illyne and Miss Hazel Maxwell, who have carried on the work in this laboratory.

In conclusion I should like to emphasize that combined clinics in diseases of systems requiring various types of therapy give the best results from the standpoint of therapy, teaching and research.

HEMOLYTIC JAUNDICE

*Its Diagnosis, Behavior and Treatment**

W. P. THOMPSON

TWO AND a half years ago we presented here a paper of this same title.[1] Tonight we are to repeat much of what was said then, but in the light of considerable further experience.

Our methods of study remain unaltered. Our patients continue to be followed at regular intervals by the combined Medical and Surgical Spleen Clinic group. Laboratory procedures continue to be done by the Spleen Clinic technicians and our central files expand rapidly.

The term "hemolytic jaundice" is still used for the general diagnosis of all patients presenting jaundice and anemia due to increased red blood cell destruction. There is, in addition, almost always an associated splenomegaly and an increase in the number of reticulocytes in the peripheral blood.

The term "hemolytic jaundice" is still being divided into two clinical subdivisions. The first of these is a uniform, recognizable, curable entity. This disease, formerly known as congenital hemolytic jaundice, is now known, at the suggestion of Dr. E. B. Krumbhaar[2], as spherocytic jaundice, a term which is far more accurate. The second subdivision consists of a heterogeneous variety of disturbances grouped together under the intentionally vague term—atypical hemolytic anemia.

SPHEROCYTIC JAUNDICE

Spherocytic jaundice is in our experience a uniform and readily recognizable disease entity provided certain diagnostic criteria are rigidly maintained. It is a chronic disease of long duration and of relative mildness. Acute exacerbations may occur but these episodes are not common. The presenting symptom is chronic, variable jaundice and

* From the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Presbyterian Hospital, New York City
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TABLE I

TYPICAL HEMOLYTIC JAUNDICE—NONSPLENECTOMY*

Case	Age	FIRST BLOOD COUNT							Duration of Jaundice Years	Spleen	Results As of October 1938
		Date	Hemoglobin, Per Cent	Red Blood Cells, Millions	White Blood Cells	Neutrophils Per Cent	Reticulocytes, Per Cent	Fragility	Microcytes, Per Cent		
1	56	2/26/22	35	2.8	5,000	77	Increased	0.55	Present	0.5	6 cm. below costal margin	Died 6/3/22, cause unknown
2	8 mo.	3/ 2/28	26	2.4	35,400	22	10.0	0.60	?	0.6	8 cm. below costal margin	Left against advice
3	50	11/15/29	68	3.2	5,300	59	15.5	0.525	Present	30	Enlarged	Symptoms continue, 39 yrs.
4	25	6/ 8/31	41	3.1	6,700	70	24.0	0.675	33	20	26 cm. below costal margin	Symptoms continue, 27 yrs.
5	18	7/14/31	78	4.1	9,000	66	10.6	0.575	Present	17	2 cm. below costal margin	Symptoms continue, 24 yrs.
6	14	8/ 2/33	55	2.7	6,400	68	12.4	0.625	15	5	1 cm. below costal margin	Symptoms continue, 9 yrs.
7	57	7/ 3/35	57	3.4	5,000	40	10.9	0.625	11	25	To umbilicus	Symptoms continue, 23 yrs.
8	7	9/25/35	68	4.1	13,200	61	17.8	0.65	7	4	To umbilicus	Symptoms continue, 7 yrs.
9	7	9/25/35	74	3.6	12,200	43	13.6	0.65	9	4	7 cm. below costal margin	Symptoms continue, 7 yrs.
10	9	9/25/35	60	3.3	7,900	51	14.5	0.65	12	5	8 cm. below costal margin	Symptoms continue, 8 yrs.
11	29	9/18/35	85	4.0	8,400	77	12.0	0.65	16	9	9.5 cm. below costal margin	Symptoms continue, 11 yrs.
12	37	6/17/36	90	3.8	6,900	58	18.8	0.60	10	37	8 cm. below costal margin	Symptoms continue, 39 yrs.
13	30	5/10/38	76	3.2	12,500	59	23.6	0.65	13	30	9 cm. below costal margin	Symptoms continue, 30 yrs.

*All patients had anemia and were jaundiced.

the outstanding physical finding is splenomegaly. The positive diagnosis is made in the laboratory. In all cases of spherocytic jaundice, the peripheral blood contains the spherical red cells that we believe are as pathognomonic of this disease as are the sickle cells of sickle cell anemia.

The Spleen Clinic has now had the opportunity of studying forty-three cases of this typical disease. Of these forty-three, thirty have had their spleens removed. The more important clinical and laboratory data are presented in the accompanying Tables I and II.

Atypical Hemolytic Anemias

In marked contrast to spherocytic jaundice is our experience with the atypical hemolytic anemias. In the former group a definite diagnosis is readily available; a prognosis as to the behavior of the disease may be made with considerable assurance; and the results of splenectomy can be predicted with security.

The atypical group comprises a total of fifteen cases presenting chronic acholuric jaundice, anemia with evidence of regeneration, and splenomegaly. In all of this group the possibility of spherocytic jaundice had to be considered but in all of these, diagnosis of spherocytic jaundice could not be sustained because the blood failed to show the spherical cells with their attendant fragility changes.

Although the total number of patients included in this group is only slightly more than we reported two and a half years previously, our experience has increased enormously. We have now had the opportunity of studying three separate family groups in which hemolytic anemia and jaundice were present in various generations and in which the diagnosis of spherocytic jaundice might well have been made, except for the absence of spherocytes. And it is our present conception that a familial type of hemolytic anemia does occur, that is not spherocytic jaundice, that is not sickle cell anemia, and that is not the erythroblastic anemia described by Cooley. What the nature of this disturbance may be we do not know; but we do know, after having removed several spleens, that splenectomy in no way alters the disease process and the splenic histology is not that so characteristic of the spherocytic type[3].

During the past two and a half years there have been several examples of a subacute to acute hemolytic anemia with jaundice seen in association with sulfanilamide medication, as well as several instances of the disturbance described by Lederer.

TABLE II

TYPICAL HEMOLYTIC JAUNDICE—SPLENECTOMY*

Case	Age	Date	Hemoglobin, Per Cent	Red Blood Cells, Millions	White Blood Cells	Neutrophils Per Cent	Reticulocytes, Per Cent	Fragility	Microcytes, Per Cent	Duration of Jaundice	Splenectomy	Spleen	Pathology of Spleen	Pathology of Gallbladder	Duration of Follow Up	Results of Operation
14	18									15 yrs.	4/10/20	Enlarged	Typical	Stones	18½yrs.	Excellent
First blood count		6/21/05	70	3.5	4,500	48			Many							
Last blood count		3/16/36	104	5.0			1.1	0.475	13							
15	14									18 yrs.	8/14/22	720 Gm.	Typical	No stones	16 yrs.	Excellent
First blood count		7/11/12	50	2.7	10,000	45		0.5+	Present							
Last blood count		6/ 2/37	100	5.3	14,500	51	2.6	.625	14							
16	4									2 yrs.	9/28/25	Enlarged	Typical	?	6 yrs.	Died August 1931, of poliomyelitis
First blood count		11/23/23	70	4.3	13,400	63	6.0	0.60								
Last blood count		12/12/29	80	5.2				0.52								
17	35									5 yrs.	6/ 4/26	1.032 Gm.	Typical	Stones		Died 3 days postoperative
First blood count		6/ 2/26	70	3.7	5,400	66		0.55								
Last blood count																
18	28									15 yrs.	11/ 8/29	675 Gm.	Typical	No stones	8½ yrs.	Excellent
First blood count		11/ 3/29	60	5.1	7,300	63	0.9	0.425								
Last blood count																
19	25									6 yrs.	6/23/25	475 Gm.	Typical	No stones	13 yrs.	Excellent
First blood count		9/ 8/24	75	3.8	8,100	74	15.0	0.58	Present							
Last blood count		4/15/36	94	5.1			2.1	0.625	12							
20	8									1 yr.	?/ ?/30			?	?	Record incomplete
First blood count		8/ 4/30	65	3.0	8,000	72	9.4	0.525	Present							
Last blood count																
21	45									11 yrs.	10/23/30	970 Gm.	Typical	No stones	3.5 yrs.	Died November 1934, Carcinoma of rectum
First blood count		10/14/30	55	3.6	10,000	78	3.0	0.60	Present							
Last blood count		2/21/34	85	3.6			4.3	0.575	Present							
22	26									5 yrs.	6/15/31	1,460 Gm.	Typical	Stones	7 yrs.	Excellent
First blood count		4/23/31	42	2.4	8,900	67	15.4	0.575	Many							
Last blood count		5/ 6/36	109	4.9			2.7	0.525	11							
23	20									19 yrs.	?/ ?/29			?	3 yrs.	Excellent
First blood count																
Last blood count		3/10/32	98	4.9			0.6	0.575	Present							
24	59									15 yrs.	3/ 1/32	1,435 Gm.	Typical	No stones	1 yr.	Died 3/5/33, Multiple sarcomatosis
First blood count		2/14/32	40	1.7	7,400	72	15.7	0.55	20							
Last blood count		6/ 8/32	82	4.7			4.0	0.525	18							
25	28									8 mos.	2/ 2/33	265 Gm.	Typical	No stones	5 yrs.	Excellent
First blood count		1/13/33	52	2.5	11,000	67	33.6	0.625	17							
Last blood count		5/ 5/34		4.4					17							
26	48									5 yrs.	?/ ?/27	Enlarged		?	7 yrs.	Excellent
First blood count		?/ ?/25	35						Present							
Last blood count		3/21/34	96	5.3			2.7	0.50	8							
27	30									9 days	3/19/34	750 Gm.	Typical	No stones	4 yrs.	Excellent
First blood count		3/ 1/34	45	1.5	21,300	76	78.6	0.625	24							
Last blood count		3/ 2/38	96	5.1	16,500	60	8.7	.60	Present							
28	35									10 yrs.	5/25/34	1,000 Gm.	Typical	No stones	4 yrs.	Excellent
First blood count									Present							
Last blood count		4/12/37	90	4.7	16,100	56	2.5		9							

Case	Age	Date	Hemoglobin, Per Cent	Red Blood Cells, Millions	White Blood Cells	Neutrophils Per Cent	Reticulocytes, Per Cent	Fragility	Microcytes, Per Cent	Duration of Jaundice	Splenectomy	Spleen	Pathology of Spleen	Pathology of Gall-bladder	Duration of Follow Up	Results of Operation
28	35									10 yrs.	5/25/34	1,000 Gm.	Typical	No stones	4 yrs.	Excellent
First blood count									Present							
Last blood count		4/12/37	90	4.7	16,100	56	2.5		9							
29	26									4 yrs.	6/27/35	600 Gm.	Typical	No stones	3 yrs.	Excellent
First blood count		5/15/35	90	4.6	6,300	64	10.7	0.625	9							
Last blood count		3/ 9/38	103	4.6	5,400	47	1.8									
30	28									18 yrs.	12/ 5/35	780 Gm.	Typical	No stones	2½ yrs.	Excellent
First blood count		11/18/35	83	3.7	17,300	79	17.6	0.70	15							
Last blood count		7/20/38	16.0 g.	4.9	12,100	75	0.7		8							
31	11									11 yrs.	11/27/33	685 Gm.	Typical	No stones	4½ yrs.	Excellent
First blood count		11/16/33	70	3.5	12,950	70	21.5	0.475	Present							
Last blood count		6/ 2/37	98	5.3	11,500	47	3.8									
32	11									3 yrs.	2/ 1/35	1,180 Gm.	Typical	No stones	3½ yrs.	Excellent
First blood count		12/14/34	53	2.6	10,600	59	20.2	.575	Many							
Last blood count		3/ 3/37	107	5.1	15,000	54	4.2	.60	11.4							
33	31									20 yrs.	11/ 5/36	2,000 Gm.	Typical	No stones	2 yrs.	Excellent
First blood count		12/ 5/34	96	4.5	18,700	86	11.2	.60	12							
Last blood count		3/14/38	122	5.1	9,000	64	2.6									
34	22									20 yrs.	5/14/36	509.6 Gm.	Typical	Stones	2 yrs.	Excellent
First blood count		4/13/36	64	3.0	7,700	74	18.9	.625	15							
Last blood count		6/29/36	90	4.5	7,650	57	0.4									
35	16									16 yrs.	8/29/36	910 Gm.	Typical	No stones	2 yrs.	Excellent
First blood count		8/13/36	45	2.4	5,400	58	45.6	.65	10.9							
Last blood count		1/ 5/38	98	4.7	12,500	68	1.5		12.0							
36	48									48 yrs.	12/ 2/36	1,311 Gm	Typical	No stones	1½ yrs.	Excellent
First blood count		8/26/36	88	3.5	6,700	61	27.3	.65	8.2							
Last blood count		6/15/38	15.8 g.	4.4	10,800	59	1.1	.525	8.8							
37	29									3 yrs.	5/ 6/37	600 Gm.	Typical	Numerous	1½ yrs.	Excellent
First blood count		1/ 6/37	86	4.0	8,700	71	16.9	.65	8.4							
Last blood count		12/15/37	102	5.4	10,100	61	1.5	.60	10							
38	36									3 yrs.	5/24/37	1,200 Gm.	Typical	Normal	1 yr.	Excellent
First blood count		5/21/37	80	3.6	9,000	59	10.6	.60	7.8							
Last blood count		5/25/38	16.2 g.	5.2	11,600	50	1.2		9.7							
39	20									20 yrs.	7/23/37	760 Gm.	Typical	No stones	1 yr.	Excellent
First blood count		7/20/37	60	3.2	7,400	58	23.9	.625	19							
Last blood count		8/ 3/38	14.5 g.	4.8	10,800	56	0.2	.50								
40	25									25 yrs.	1/14/38	860 Gm.	Typical	No stones	8 mos.	Excellent
First blood count		10/20/37	78	3.4	12,000	80	10.8	.625	10							
Last blood count		8/ 3/38	17.0 g.	4.7	14,100	60	2.6	.400								
41	15									2 mos.	12/ 8/35	Enlarged	Typical	No stones	2½ yrs.	Excellent
First blood count		11/ 1/38	54	3.75	6,200		10	Increased	Many							
Last blood count		6/ 8/38	15.7 g.	5.4	23,500	91	1.3	.525	76							
42	36									36 yrs.	7/20/38	330 Gm.	Typical	Pigment. stone	2 mos.	Died 10/10/38 Multiple
First blood count		6/20/38	7.8 g.	2.1	14,800	68	50.9	.65	15.6							
Last blood count		10/ 3/38	4.5 g.	1.1	20,800	60	78									Accessory Spleens
43	35									35 yrs.	8/ 9/38	900 Gm.	Typical	No stones	3 days	Died 8/12/38 Pneumonia
First blood count		7/21/38	4.6 g.	1.25	6,100	79	25.1	.525	19							
Last blood count		8/12/38	8.6 g.	2.7	28,000	98	34.1									

TABLE III

ATYPICAL HEMOLYTIC ANEMIA*

Case	Age	FIRST BLOOD COUNT							Duration of Symptoms	Splenectomy	Spleen	Pathology of Spleen	Pathology of Gallbladder	Duration of Follow Up	Results
		Date	Hemoglobin, Per Cent	Red Blood Cells, Millions	White Blood Cells	Neutrophils Per Cent	Reticulocytes, Per Cent	Fragility	Microcytes, Per Cent							
Cases in which splenectomy was performed															
44	24	3/ 1/20	50	4.7	6,700	62	1.0	0.55	0	4.5 yrs.	12/ 6/24	397 Gm.	Normal	No stones	14 yrs.	Improved; diagnosis unknown
45	27	7/31/29	20	0.94	31,200	80	19.0	0.425	0	4 days	10/25/29	540 Gm.	Marked blood formation	Stones	1 yr.	Died 10/4/30; autopsy; acute course partly arrested; diagnosis unknown
46	63	9/19/31	35	1.3	3,300	78	37.0	0.425	0	4 wks.	10/31/31	680 Gm.	Marked blood formation	No stones	1 mo.	Died 12/5/31; autopsy; Splenectomy no help; diagnosis unknown
47	16	6/23/33	80	3.9	10,000	59	5.7	0.425	0	11 yrs.	6/28/35	540 Gm.	Not typical	No stones	3 yrs.	Situation 3 yrs. post-op. unchanged
48	1½	9/16/33	38	1.6	4,300	39	13.2	.475	0	1½ yrs.	9/21/33	225 Gm.	Normal	Normal	5 yrs.	Slightly improved
49	2 mo.	2/ 4/37	39	1.7	7,700	32	2.6	.525	0	2 mos.	4/16/38	225 Gm.	Normal	Normal	5 mos.	Slightly improved
50	21	8/ 3/37	82	3.3	7,800	74	39.0	.45	0	21 yrs.	8/21/37	370 Gm.	Sl. Fibrosis	Pigment. stone	None	Died; Post-op. shock
Cases in which splenectomy was not performed															
51	44	8/ 4/28	15	0.61	10,400	74	25.0	.....	0	6 mos.	None	580 Gm.	Sarcoma	.....	.....	Died 8/6/28; autopsy; sarcoma of spleen
52	56	9/20/29	35	1.5	7,700	88	6.4	0.425	0	2 mos.	None	1,300 Gm.	Sarcoma	Normal	.....	Died 10/23/29; autopsy; sarcoma of spleen
53	72	1/15/32	48	1.8	6,500	76	27.2	0.425	0	6 mos.	None	820 Gm.	Sarcoma	.....	.....	Died 5/23/35; autopsy; radiotherapy helped for a time; sarcoma of spleen
54	52	8/ 4/30	33	1.3	2,300	73	....	Normal	0	3 wks.	None	.....	.....	.....	4½ yrs.	Died—Syphilis of spleen
55	64	3/ 3/32	64	3.1	3,200	44	10.2	0.45	0	2 yrs.	None	Palpable	.....	.....	.....	Died 7/5/32; apparently cerebral accident; diagnosis unknown
56	21	4/ 5/33	108	6.1	15,700	74	4.6	0.50	0	4 yrs.	None	3 cm. below costal marg.	.....	.....	.....	No change, 1/8/35; diagnosis unknown
57	80	11/30/36	32	1.4	25,000	88	39.5	Normal	0	1 yr.	None	310 Gm.	Sarcoma	.....	2 mos.	Died—Retic. Cell sarcoma
58	60	4/13/37	58	1.9	5,300	60	20.3	Normal	0	1½ yrs.	None	.....	.....	.....	3 yrs.	Condition unchanged.

* All patients had anemia (except patient 56) and all were jaundiced.

Otherwise, our group of atypical hemolytic anemias remains as previously reported. Details of this group are available in the accompanying Table III.

The Nature of Spherocytic Jaundice

The clinical study of patients with spherocytic jaundice, before, during and after operation, has led to certain conclusions concerning its behavior. The symptoms of chronic anemia with jaundice may appear at any age or an enlarged spleen may be found in an individual being examined for some unrelated complaint. The anemia may be mild or severe; or, in the latent cases, the blood count may be quite normal. The degree of jaundice varies likewise but is always proportional to the degree of anemia. The percentage of reticulocytes also varies within wide limits, depending again on the severity of the hemolytic process.

In every case of spherocytic jaundice the peripheral blood smear contains erythrocytes the diameter of which is obviously much less than normal but which in addition show no central pallor. In wet preparations these small cells appear spherical. With the micromanipulator these cells can be slowly and carefully examined and their spherical shape confirmed.

It is important that these cells should be detected and counted in wet film preparations with, when possible, the aid of the micromanipulator. Techniques that give the mean corpuscular volume may be misleading, as these spherical cells form only a small proportion of the total red cell colony, and many of the remaining cells, the reticulocytes, may be considerably larger than normal. In the presence of a high degree of reticulocytosis, mean corpuscular volume readings may be on the high side of normal and the spherical cells remain undetected.

It was not until the recent work of Haden[4] appeared that the relationship between shape, size and hemolysis was conclusively proved. His work leaves no doubt about the fact that these spherical cells are alone responsible for the fragility changes seen in all cases of spherocytic jaundice.

In addition, evidence is available to suggest a serious consideration of the importance of these spherical cells in the production of the active disease process.

This chain of evidence, although fragmentary in places, may be presented in order:

* All patients had anemia (except patient 56) and all were jaundiced.

1. The fact may be accepted that all patients with spherocytic jaundice, as well as their relatives with the latent disease, have these spherical microcytes circulating in the peripheral blood. These abnormal cells constitute from 10 to 25 per cent of the total number of erythrocytes, the remaining cells being either reticulocytes or normal red cells. We have never observed reticulation in the spherical cells.

2. These spherical cells are directly responsible for the fragility changes to hypotonic salt solution.

3. These spherical cells appear to be selectively removed from the general circulation of the spleen, where they are found in larger numbers than in the peripheral blood.

4. There is enough evidence, I believe, to warrant the assumption that all the increased red blood cell destruction that results in the active phase of the disease takes place within the spleen. Some of this evidence is direct; most of it is inferred.

Direct evidence can be procured only by measuring the serum bilirubin concentrations in the blood of the splenic artery and vein at the time of operation. Obvious technical difficulties interfere with the collection of the necessary specimens. In spite of these difficulties we have found that the blood of the splenic vein contains more pigment than does the blood of the splenic artery.

These observations are in accord with the figures obtained by Rich and Rienhoff[5] in an active case of hemolytic jaundice and show that the spleen is actively destroying erythrocytes.

Indirect evidence concerning the activity of the spleen in this disease may be procured by observing the effects of splenectomy. Our therapeutic results in the thirty cases in which splenectomy was performed are uniform and coincide with the results of others. Removal of the spleen is immediately followed by cessation of the increased hemolytic activity. Relief is immediate and, apparently, permanent.

A study of the factors that might be influenced by splenectomy, results in the acceptance of only one; the excessive hemolysis stops. I can see no reason to consider any other influence, as the cessation of excessive hemolysis will explain all the changes observed.

With the removal of the site of increased destruction of erythrocytes, the serum bilirubin and the urobilin excretion figures drop promptly to normal levels. The already overactive bone marrow rapidly raises the erythrocyte concentration in the peripheral blood. As the red blood cell

count rises, the stimulus for new erythrocytes falls and the proportion of reticulocytes drops. All these changes toward normal appear within a few hours after operation and are concluded in from two to three weeks. The results of the operation appear to be permanent.

Only one patient has failed to show these clear cut results. This patient, a young woman, was admitted with spherocytic jaundice, of relatively brief duration, of considerable intensity. The history, physical findings and laboratory data were all complete and unquestioned. In July of this year, her spleen was removed and the spleen sections were typical in all respects. Following splenectomy the disease process appeared to subside for the first few days but then reappeared with renewed vigor. During the next three months, spherocytic jaundice in its active phase continued in spite of multiple transfusions, the anemia increased and the reticulocyte level rose to above 75 per cent. A report of a similar instance appeared during the summer from England[6]. In the British case, the second exploratory laparotomy, done with the hope of finding an accessory spleen, revealed an ovarian tumor which was subsequently found to be a teratoma. Removal of this tumor was immediately followed by cessation of the hemolytic activity and a subsidence of all symptoms. We were unable, in our patient, to find any abnormalities on pelvic examination and the Aschheim-Zondek test was negative. Finally, it was decided that additional spleens must be present and that a second operation had to be performed. Two days after this decision was reached, an acute hemolytic crisis set in, the patient's red count fell rapidly to 600,000, the reticulocytes rose to 90 per cent and the patient died of acute spherocytic jaundice. At autopsy, the left upper quadrant contained many accessory spleens, varying in size from 1 cm. to 6-8 cm. in diameter and these accessory spleens presented the typical histology of this disease process. Our one failure, then, seems to have been a result of a congenital abnormality.

The only other theoretically possible effect of splenectomy would be some change in the spherical cells and their fragility. That some such change does occur has been suggested at various times by various authors. Their conclusions are, as a rule, variable and the changes usually observed are slight shifts in the fragility test.

Our observations warrant the definite conclusions, in the post-splenectomy cases, that the spherical cells persist with their attendant fragility changes for as long after splenectomy as time has permitted

observation. The proportion of these typical cells may vary within relatively narrow limits but they have been demonstrated in patients sixteen years after operation in concentrations as high as 14 per cent.

The recent report of Lord Dawson of Penn[7] of typical fragility changes persisting forty-five years after splenectomy leaves little doubt that the spherical cells are not affected by this surgical procedure.

This evidence, I feel, warrants the conclusions that, given the inherited spherical microcytes and increased hemolytic activity, one has the condition known as spherocytic jaundice, and that this disease, once active, remains active until the site of increased hemolytic activity is removed.

Three important questions still remain unsolved.

1. Little or nothing is known about the genetics of transmission of these spherical cells.

2. Little or nothing is known about the factors that initiate the excessive hemolysis which signals the onset of the active disease. It is known that individuals exist, as members of spherocytic families, whose blood contains these spherical cells and in whom increased hemolysis is not taking place. We also know that these latent cases may become active at any time in their career from, in our series, the age of six weeks to the age of fifty-eight years. We also know that when the disease once becomes active, spontaneous remission will not occur and that chronic anemia with jaundice will persist until splenectomy. But we have not been able to associate the change from latency to activity with any as yet discernible event. Occasionally an acute infection precedes the onset of jaundice; in the majority of cases no such history can be elicited.

3. We do not know whether a normal spleen interposed in a circulation containing spherical erythrocytes would behave as and assume the histologic characteristics of a spleen presenting spherocytic jaundice.

Conclusions

1. Hemolytic anemia with jaundice and splenomegaly has been observed in fifty-five patients.

2. This syndrome may be separated into two groups:

   (a) Spherocytic jaundice

   (b) Atypical hemolytic anemia

3. Spherocytic jaundice is a clear cut, definite disease entity the course of which can be predicted and the mechanism of which is begin-

ning to be understood.

4. The atypical hemolytic anemias comprise a heterogeneous group of conditions in which there is increased red cell destruction.

5. The symptoms of spherocytic jaundice are promptly, completely, and permanently relieved by splenectomy. The atypical hemolytic anemias have not been benefited by this procedure.

6. Accurate preoperative diagnosis is one of great importance and a familial incidence is of no differential value.

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CONGESTIVE SPLENOMEGALY*
(BANTI'S SYNDROME)

LOUIS M. ROUSSELOT

Two years ago a report[1] was presented from the Spleen Clinic of the Presbyterian Hospital outlining the results of splenectomy in Banti's syndrome. At that time we stressed the role of venous congestion or portal stasis in the production of the syndrome.

In this paper we wish to re-emphasize the same hypothesis and present additional evidence to support our contention that Banti's syndrome is not a primary splenomegaly, but rather a splenomegaly secondary to portal stasis. We have come to consider this group of splenomegalies under the general caption of "Congestive Splenomegaly," as this we feel more aptly describes the condition. The term "Congestive Splenomegaly" implies a primary congestive mechanism in the portal bed producing back pressure (splenic or portal vein hypertension). The venous stasis we believe subsequently produces a splenomegaly and certain other characteristic clinical and laboratory findings. This hypothesis is, of course, a reversal of Banti's[2,3] original idea that the disease began as a primary splenomegaly of toxic origin and later developed into cirrhosis of the liver. The conception of Banti's syndrome as a congestive splenomegaly and not a primary splenomegaly is not a new one. Warthin[4], Eppinger[5], and Larrabee[6], all considered the possibility of portal congestion producing the Banti picture. Eppinger, in fact, frequently refers to the "Stauungsmilz" in his treatise on hepatolienal fibrosis.

McMichael[7] first suggested the term "Portal Hypertension." Both he and McNee[8] agreed that the factor of increased portal pressure plus the entry of toxic substances into the portal circulation probably accounted for the development of Banti's disease. McMichael likewise intimated that a condition of portal hypertension can occur in the absence of such gross changes as are found in a hobnail liver. This latter

* From the Department of Surgery, Columbia University, College of Physicians and Surgeons and the Spleen Clinic of the Presbyterian Hospital. Delivered November 4, 1938 in the Eleventh Annual Graduate Fortnight

statement is significant, for we have been able to demonstrate conclusively portal hypertension in many cases without cirrhosis. Portal hypertension we believe is usually secondary to one of a variety of totally unrelated diseases. Any of these conditions is capable of initiating chronic portal obstruction. Our own experience as to the types of different primary lesions which may produce congestive splenomegaly will be discussed in due course. The distended and tense venous radicals in the splenic pedicle, and the rich venous collateral often enveloping the spleen, are visible phenomena that never fail to attract anyone familiar with surgery of the spleen in congestive splenomegaly. This local vascular condition occurs regardless of the nature of the obstructive factor in any particular case. Believing that portal hypertension may be a common factor associated with a variety of disturbances producing congestive splenomegaly, members of our Clinic[9] recently have made observations on the splenic vein pressures at operation. These studies are being continued. The determinations are made at the time of operation, after mobilization of the spleen, by inserting the needle of a venous pressure apparatus into the splenic vein. Simultaneously a reading is taken of the peripheral venous pressure in one of the veins of the arm.

Splenic vein pressure readings have now been made in fourteen cases of congestive splenomegaly or Banti's syndrome, and in fifteen controls having splenic lesions of other types. In the accompanying table these figures are listed in the various sub-groupings under which we now classify our various cases.

An obstructive factor as a cause for the portal hypertension was present in 52 per cent of our early cases and no such factor was ascertainable in 48 per cent of the same group. We have found that the 48 per cent group with no obvious obstructive factor comprises chiefly the portal bed and a liver biopsy were not part of the operative our oldest cases, treated at a time when a systematic exploration of routine. However, in the past two years we have done splenectomies on twenty-four patients in addition to the previously reported thirty-one cases. With more careful inspection of the portal bed and liver biopsy on all the recent cases, and the securing of a post-mortem examination on several of the long-term cases in which an obstructive factor had previously been unknown to us, the percentage of "unknowns" is now diminishing. In these last twenty-four cases, therefore, seventeen or

Table I

VENOUS PRESSURES IN MILLIMETERS OF NORMAL SALINE

BANTI—LAENNEC CIRRHOSIS

	Splenic Vein	*Arm Vein*
C.M. 1.	225	12
G.M. 2.	325	85
D.P. 3.	450	125
N.A. 4.	470	145
R.B. 5.	370	30

BANTI—SCHISTOSOMIASIS MANSONI

	Splenic Vein	*Arm Vein*
P.R. 1.	250	50
A.E. 2.	335	105
G.P. 3.	500+	70
C.C. 4.	415	125
L.M. 5.	375	60

BANTI—THROMBOSIS OF SPLENIC VEIN

	Splenic Vein	*Arm Vein*
J.S. 1.	390	170

BANTI—OBSTRUCTIVE FACTOR UNDETERMINED

	Splenic Vein	*Arm Vein*
L.D. 1.	275	105 (p adrenalin)
G.K. 2.	370	50
B.S. 3.	330	55

CONTROLS

	Splenic Vein	*Arm Vein*	*Diagnosis*
F.H. 1.	190	65	Lymphosarcoma
R.B. 2.	105	80	Hemolytic Jaundice
L.L. 3.	220	205	Atyp. Hemol. Jaundice
N.B. 4.	125	130	Hemolytic Jaundice
S.S. 5.	215	40	Gaucher's
L.L. 6.	190	107	Splen. Undet. Origin
W.U. 7.	120	95	Hemolytic Jaundice
S.J. 8.	360	5 (severe shock)	Purpura
Z.G. 9.	185	75 (beginning shock)	Pancreatic Adenoma
S.L. 10.	190	210	Purpura
G.C. 11.	235	165	Splen. Undet. Origin
E.N. 12.	275	205	Atyp. Hemol. Jaundice
T.K. 13.	70	300 (10 min. later)	Purpura
M.M. 14.	140	65	Purpura
J.K. 15.	245	240	Lymphatic Leukemia

70 per cent have had a definite obstructive factor.

The number of cases of congestive splenomegaly studied by the Spleen Clinic that have had splenectomy now totals fifty-five. Sixty per cent (thirty-three cases) of the entire group have had a proven obstructive factor as a possible basis for the portal hypertension.

Under the general classification of congestive splenomegaly we have continued to use the following groupings and sub-groupings:

I. Obstructive factor known.
   A. Cirrhosis of the liver.
      a. Laennec's cirrhosis.
      b. Unclassified cirrhosis.
      c. Cirrhosis due to schistosomiasis mansoni.
   B. Thrombosis of the splenic vein.
   C. Cavernomatous transformation of the portal vein.
   D. Stenosis of the portal vein.

II. Obstructive factor undetermined.

The results of splenectomy and the long-term follow-up will now be detailed group by group under the separate disease headings (Charts I and II) as just outlined. These cases have regularly been examined and complete blood studies made at least once a year since operation.

I. Obstructive factor known.
   A. Cirrhosis of the liver.
      a. Laennec's cirrhosis:

Fourteen cases of this type were operated upon. The immediate hospital mortality included four cases or 28 per cent. The causes of death were: shock (1); hematemesis (1); hepatic insufficiency (2).

Seven late fatalities occurred at periods varying from five months to thirteen years. Death in all cases followed hematemesis, or cholemia with or without ascites.

The three survivors have been well to date for periods of one and a half years, five years, and six years, respectively. These poor results are at distinct variance with those found in the succeeding groups.

      b. Unclassified cirrhosis:

Two cases are included in this grouping; both have done exceptionally well, one for three years and one for twelve years following operation.

CONGESTIVE SPLENOMEGALY
(BANTI'S SYNDROME)
RESULTS OF SPLENECTOMY

Chart I

1. Each circle represents six months in the clinical course.
2. The letter S indicates time of splenectomy during the entire period patient was followed.
3. Clear circles denote asymptomatic periods. Shaded circles denote periods with symptoms. Black circles denote death.

Chart II (refer to key under Chart I)

c. Cirrhosis due to schistosomiasis mansoni*:
Eleven cases of this disease with associated Banti's syndrome have had splenectomy. The mode of infection and the pathology of schistosomiasis are now well known. Faust, Jones, and Hoffman[10] have shown that the maturing phase of the parasite in the human host takes place in the hepatic,

portal and mesenteric veins. Following the deposition of eggs in the liver, a pseudo-tubercle or fibrous nodule is formed. If the infestation is sufficiently heavy, a severe form of cirrhosis results. Coupled with this cirrhosis is a splenomegaly, and with the splenomegaly there is often a blood picture similar to that in Banti's syndrome. Oddly enough, the striking similarity of Banti's disease to the late visceral stage of schistosomiasis mansoni is one that has received scant mention. Bonelli[11] drew the clinical comparison but concluded that the Banti-like picture in late schistosomiasis was on the basis of a toxin of intestinal origin. Girges[12] with his extensive experience in Egyptian splenomegaly includes Banti's disease as a differential diagnosis of the late stage of the disease. A particularly interesting feature of our experience in these cases is that prior to operation in several of them a clinical diagnosis of Banti's disease was made and only following liver biopsy was the exact nature of the lesion ascertained. Campbell[13] in a recent publication from China has expressed the belief that most cases of Banti's disease in the Orient are really the late stage of schistosomiasis.

There has been no operative mortality in our eleven cases. The only patient who had hematemeses prior to operation remained well for a period of two and a half years after operation and then suddenly succumbed to a hematemesis. All the remaining ten patients are very well and have been followed for periods varying from two to four years.

B. Thrombosis of the splenic vein.

We have had three cases of Banti's syndrome associated with splenic vein thrombosis. All had an uneventful postoperative recovery and are well at ten months, five and a half years, and six years, respectively.

C. Cavernomatous transformation of the portal vein.

We have had two examples of this rare condition. Both cases had the typical clinical picture of Banti's syndrome with evidence of bleeding from esophageal varices. In each instance hematemesis was the cause of death following operation – in the first case, two days after operation, and in the second instance,

* A more detailed and complete report of the association of Banti's syndrome in schistosomiasis and the results of splenectomy in this disease, is in press.

nine months after operation. Of particular interest to us is the fact that the liver was normal in each of these cases, proven by autopsy; hence it is obvious that hematemesis is not necessarily the sequel of cirrhosis but can be a fatal issue in the presence of obstructive lesions other than cirrhosis. One of these cases was particularly instructive to us because prior to operation all the liver function tests gave normal values; at operation the liver grossly appeared normal and the liver biopsy likewise showed normal liver structure. We therefore classified this as one of the cases in which the obstructive factor was undetermined. It was only when the patient died nine months later that autopsy demonstrated the obstructive factor at the beginning of the portal vein. Klemperer[14] reported one such case and was able to collect twenty-three similar examples in the literature. His patient, as ours, also had undergone a splenectomy for supposed Banti's disease several years prior to death.

D. Stenosis of the portal vein.

This is a recent addition to our list of possible lesions causing portal congestion. We have had one patient with this unusual condition. At operation no obstructive factor was demonstrated. The liver biopsy was normal. Following operation the patient had six hematemeses over a period of thirteen years, with finally a fatal hemorrhage. At necropsy there was no cirrhosis, but a stenosis of the portal vein was present near its origin.

II. Obstructive factor, undetermined.

Our last series in the congestive splenomegaly group includes twenty-two cases in which the obstructive factor could not be demonstrated at operation. Three died in the postoperative period, a mortality of 13.6 per cent. The cause of death included shock (1); hepatic insufficiency (2).

Only one late death occurred, and this, two years and two months after operation, of hematemesis.

Two died several years after operation of an unrelated disease. Of the remaining sixteen, thirteen have been entirely well for periods varying from two months to eighteen years. However, there are three patients still living who have had a miserable course punctuated by repeated, severe hematemesis over periods of three years, four years, and two and a half years, respectively.

Summary

In a group of fifty-five cases, similar characteristics were apparent in all, to warrant a diagnosis of congestive splenomegaly, or so-called Banti's syndrome. Evidence has been presented to show that portal hypertension exists as a common factor in a variety of different clinical entities. Each of these various diseases is capable of producing chronic portal stasis.

The indications for splenectomy in congestive splenomegaly and the long term prognosis are dependent on the nature and severity of the obstructive factor. Obviously, splenectomy is contraindicated in cases of progressive decompensated liver disease. The late results in our cases with Laennec's cirrhosis, portal vein occlusion or stenosis, have been extremely poor. The results in other forms of cirrhosis, splenic vein thrombosis, and the group in which the obstructive factor was undetermined, have been most gratifying. Hematemesis as a postoperative symptom is usually a grave prognostic omen.

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DIAGNOSTIC AND THERAPEUTIC CONSIDERATIONS IN THE MANAGEMENT OF IDIOPATHIC THROMBOCYTOPENIC PURPURA*

R. H. EGERTON ELLIOTT

IN 1936, a report[1] was published from the Spleen Clinic of the Presbyterian Hospital on the results of the treatment of purpura hemorrhagica by both splenectomy and conservative therapy. The object of this present paper is to bring those results up to date and, in addition, to relate some of the salient features of the diagnosis and management of this disease as it is seen in the spleen clinic.

There are certain elementary facts about purpura in general and thrombocytopenic purpura in particular which should be constantly borne in mind in any consideration of the management of the disease under discussion. It therefore seems advisable to review briefly the more important of these facts before proceeding further.

In the vast majority of instances in which it is encountered, purpura represents a symptom-complex rather than a disease entity. The frequency of its occurrence as such is attested by the fact that it may appear in the course of many common ailments. Under these circumstances, it is referred to as a "secondary" or "symptomatic" purpura.

The term "primary" or "idiopathic" purpura is reserved for those infrequent instances in which no associated disease process or cause for the symptom-complex can be found. The relative rarity of this latter group becomes apparent when it is considered that out of approximately 700 purpuras which have passed through the spleen clinic, only forty-two have been classified as "primary" or "idiopathic."

SECONDARY PURPURA

The secondary purpuras, then, are those for which there is thought to be an assignable cause. They are the purpuras which occur with

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infections, with poisoning by toxic agents, with nutritional deficiencies and with certain disease processes affecting the reticulo-endothelial and hematopoietic systems. According to whether the platelet count is reduced or not, they may be subdivided into secondary purpuras without thrombocytopenia and secondary purpuras with thrombocytopenia.

Secondary purpura without thrombocytopenia is encountered with considerably more frequency than the thrombocytopenic variety. While it may be associated with any of the previously mentioned etiologic factors, it is most commonly seen in the course of acute infections. In most instances, its recognition as a secondary purpura is attended with little difficulty.

Secondary purpura with thrombocytopenia, on the other hand, may at times be exceedingly difficult to differentiate from a primary or idiopathic purpura. It likewise may occur in conjunction with any of the aforementioned etiologic factors, but in our experience is most frequently seen in disturbances of the hematopoietic system, in particular the aplastic anemias and the leukemias.

The important point to be remembered about the management of the secondary purpuras as a whole is that therapy should be directed primarily at the disease process underlying the purpura. That the performance of splenectomy for the relief of hemorrhagic manifestations in this group is emphatically contraindicated and often attended by tragic consequences, will be apparent when we come to discuss our results.

Primary Purpura

Bearing in mind the foregoing considerations, it will be realized that the diagnosis of primary or idiopathic thrombocytopenic purpura can only be reached by the careful exclusion of all factors known to cause purpura. Unfortunately, those disease processes which most frequently cause an associated thrombocytopenic purpura are often the most difficult of recognition, as, for example, an aplastic anemia or an aleukemic leukemia. For this reason, the *sine qua non* of diagnosis is an accurate hematologic interpretation of the blood findings.

It also goes without saying that other common pitfalls in diagnosis can only be avoided by the taking of a careful history, with especial reference to dietary habits and drug idiosyncrasies, and by an exhaustive search for foci of infection in the teeth, tonsils, sinuses and other

common locations. In this connection, it should also be borne in mind that metastatic invasion of the bone marrow from an unsuspected primary focus of malignancy may occasionally cause thrombocytopenic purpura. In our records, we have four such cases, all of them proven at necropsy.

Natural History of the Disease

Primary or idiopathic thrombocytopenic purpura may occur at any age, but in our experience is most frequently seen before the fourth decade. It is uncommon beyond the fifth decade and rare in the aged. If untreated, its course in the adult is characterized by chronicity and recurrence.

It is perhaps fitting at this point to say a word about the occurrence of this disease in children. Though the great majority of patients who attend the spleen clinic are adults, through the co-operation of the Hematology Clinic of the Babies Hospital we have of late years been privileged to see an increasing number of individuals in the early age groups. Furthermore, as these patients mature, they are automatically transferred for their future follow-up to our clinic. We have, therefore, been afforded the opportunity of obtaining unusually complete follow-ups on many of our cases.

From a perspective of the disease so obtained and from our frequent contact with the members of the hematologic group of the Babies Hospital, we have come to feel that thrombocytopenic purpura in children generally presents a different picture and a different problem from that observed in adults.

In childhood, idiopathic thrombocytopenic purpura tends to be a self-limited disease, characterized by spontaneous recovery. In relatively few instances does it persist into adulthood as a chronic, recurrent ailment. According to McLean, Kreidel and Caffey[2] it affects boys twice as frequently as girls.

In adults, on the other hand, chronicity and recurrence in the untreated case are the rule, rather than the exception. Furthermore, our figures for sex incidence would seem to indicate that the disease occurs principally in females, for, out of forty-two patients, thirty-seven or 88 per cent were women and only five, or 12 per cent, men. It is of interest that at the onset of the disease, four of the five males were under fourteen years of age and the fifth had not reached his twenties.

Clinical Picture

The disease, then, is generally seen in young females in the second or third decade of life. A variable history of easy bruising, of bleeding into the skin and of hemorrhage into, or from the mucous membranes may be given. Intractable menorrhagia is a frequent and distressing symptom. In advanced cases hematuria, melena, blurring of vision and symptoms due to cerebral hemorrhage are not uncommon.

On physical examination, evidence of hemorrhage into the skin or mucous membranes, or both, will be present to a variable extent. If the patient is in an active phase of the disease, a typical muddy pallor may be apparent. The gums are often characteristically soft, swollen and spongy, the teeth caked with clotted blood. In addition, in the far advanced disease intra-ocular hemorrhages and reflex changes may be present.

The existence of a palpable spleen is unlikely in early cases but in individuals who have had the disease over a long period of time, its tip can occasionally be appreciated just below the costal margin. In our series of forty-two cases, the organ was felt in ten instances, and in only one of these was the disease in its early stages. It should be emphasized, however, that the presence of any considerable degree of splenomegaly is a point against the diagnosis of idiopathic thrombocytopenic purpura. The remainder of the physical examination will generally be non-contributory.

Laboratory Findings

With regard to the various laboratory findings, an anemia of the secondary type is usual, but is never in excess of that due to blood loss. The white and differential blood counts are normal except in severe cases with widespread hemorrhage, when a polymorphonuclear leukocytosis may be expected. The presence of a leukopenia should cast immediate doubt upon the diagnosis. More often than not, it indicates the presence of an underlying aplastic anemia or other blood dyscrasia. The platelets, of course, are markedly reduced in number and may even be absent on direct examination.

In the classic case, the bleeding time is prolonged, the clotting time normal and the clot retraction delayed or absent. Occult or frank blood may be present in the stool, urine or gastric content. The tourniquet test is usually positive.

Of even more importance than the latter, the capillary resistance, as measured by the Dalldorf suction apparatus, will be reduced in proportion to the severity of the disease. This latter test, which was the subject of a recent communication[3] from this clinic, has in our hands proved extremely useful in estimating the activity of the disease. In this respect, we believe it to be frequently of more value than the platelet count. A summary of the majority of the aforementioned findings will be found in Tables I and II (See next page).

Pathology

At operation, some degree of enlargement of the spleen was reported in all but eight of our cases. Typically, however, the spleen is small and soft in comparison with other varieties of splenomegaly.

There is no general agreement among authors on the existence of a characteristic pathological picture in the spleens removed from individuals suffering from purpura hemorrhagica. In 1937, Nickerson and Sunderland[4] of the Mallory Institute of Pathology in Boston reported the presence of fairly constant pathologic change in the spleens from eleven cases of this disease.

They found that in every instance, the germinal centers were enlarged and active and that megakaryocytes were present in the sinuses of the pulp. Furthermore, the number of neutrophile or eosinophile polymorphonuclear leukocytes was increased in the pulp in all but two cases. These changes they regarded as characteristic of idiopathic thrombocytopenic purpura hemorrhagica.

We are not prepared to say more than that the majority of the spleens removed from our cases have shown changes consistent with these findings.

Management

Following the admission of a case of purpura to the hospital, the platelet count and capillary resistance are watched closely. An attempt is made to improve the general condition of the patient by ordinary supportive measures and, when indicated, by repeated transfusions. While these steps are being taken, a careful search for foci of infection is undertaken, consultants being used freely in the process.

After a thorough-going period of study, during which all other diagnostic possibilities have been ruled out to the satisfaction of the

TABLE I

INITIAL BLOOD PICTURE AND SEVERITY ON ADMISSION IN GROUP IN WHICH OPERATION WAS PERFORMED

Case	Severity	Platelets	Bleeding Time	Clotting Time	Clot Retraction	Capillary Resistance	Tourniquet Test	Hb % (Sahli)	Rbc	Wbc	Pmn	Lym	Other Cells
1	Moderate	20,000	5′	25′*	Prolonged	Not done	Not done	Not done	3.2	12.2	55	27	18
2	Moderate	Too few to count	9′	8′	Prolonged	Not done	Not done	75	4.1	10.3	59	24	17
3	Mild	15,000	3′ 30″	4′	Normal	Not done	Not done	53	3.3	10.9	74	23	3
4	Severe	28,000	58′	1′ 30″	Not done	Not done	Not done	21	1.9	24.0	84	10	6
5	Moderate	6,000	10′	3′	Prolonged	Not done	Positive	65	3.7	8.7	60	32	8
6	Moderate	15,000	5′ 30″	3′	Prolonged	Not done	Not done	56	3.1	9.9	73	21	6
7	Severe	6,000	7′	5′	Not done	Not done	Not done	33	3.2	5.5	85	10	5
8	Moderate	19,000	15′+	5′	Prolonged	Not done	Positive	75	5.3	8.6	52	32	16
9	Severe	15,000	4′	5′ 30″	Not done	Not done	Not done	32	2.2	5.7	65	25	10
10	Moderate	26,000	15′ 30″	2′ 45″	Prolonged	Not done	Positive	75	3.8	6.9	72	16	12
11	Severe	60,000	25′+	7′	Normal	Not done	Not done	60	2.9	8.8	64	28	8
12	Moderate	5,000	4′ 37″	3′ 20″	Prolonged	10	Positive	89	4.9	8.1	88	6	6
13	Severe	10,000	15′+	3′	Prolonged	10	Not done	55	3.0	7.5	75	20	5
14	Severe	26,000	13′ 35″	1′ 55″	Prolonged	10	Positive	102	6.2	15.7	78	18	4
15	Mild	23,000	20′+	3′	Not done	10	Not done	40	4.1	18.0	60	33	7
16	Mild	15,000	3′ 15″	4′ 8″	Not done	15	Not done	90	4.6	8.0	55	35	10
17	Moderate	20,000	20′	6′	Prolonged	20	Not done	57	4.3	6.2	66	20	14
18	Moderate	16,000	15′+	4′	Not done	10	Not done	76	3.4	12.4	72	20	8
19	Mild	10,000	Not done	Not done	Not done	15	Positive	92	4.8	6.4	51	45	4
20	Moderate	31,000	3′	2′ 30″	Prolonged	10	Not done	55	4.6	9.4	62	35	3
21	Moderate	22,000	1′	5′ 15″	Not done	10	Positive	38	2.2	7.0	78	14	8

* Accuracy of this value open to question.

TABLE II

INITIAL BLOOD PICTURE AND SEVERITY ON ADMISSION IN GROUP IN WHICH OPERATION WAS NOT PERFORMED

Case	Severity	Platelets	Bleeding Time	Clotting Time	Clot Retraction	Capillary Resistance	Tourniquet Test	Hb % (Sahli)	Rbc	Wbc	Pmn	Lym	Other Cells
1	Severe	Too few to count	36′	5′	Prolonged	Not done	Not done	28	2.7	17.5	79	19	2
2	Moderate	15,000	21′ 20″	2′ 30″	Prolonged	Not done	Positive	74	4.1	8.6	54	37	9
3	Moderate	35,000	28′ 30″	3′	Prolonged	Not done	Positive	70	3.4	9.5	59	36	5
4	Mild	45,000	15′	1′	Prolonged	Not done	Positive	100	5.8	5.5	48	50	2
5	Mild	20,000	4′ 30″	5′ 30″	Normal	Not done	Negative	45	3.1	10.0	73	19	8
6	Moderate	8,000	33′+	3′	Prolonged	Not done	Not done	78	4.4	10.5	66	22	12
7	Mild	28,000	9′ 45″	3′	Prolonged	Not done	Positive	85	3.9	8.4	78	14	8
8	Moderate	8,000	4′ 30″	2′ 15″	Not done	Not done	Positive	88	5.4	6.9	72	16	12
9	Moderate	6,000	12′	2′ 30″	Not done	Not done	Not done	63	3.9	5.8	60	26	14
10	Moderate	12,000	6′ 30″	2′ 30″	Prolonged	Not done	Not done	75	3.2	7.2	47	48	5
11	Mild	40,000	3′ 50″	5′ 45″	Prolonged	Not done	Not done	76	4.4	5.3	65	34	1
12	Mild	20,000	4′	Not done	Normal	Not done	Negative	85	4.7	8.6	62	34	4
13	Mild	10,000	11′ 45″	8′	Not done	20	Positive	100	4.8	5.7	63	34	3
14	Mild	6,000	5′ 30″	2′ 25″	Prolonged	15	Positive	85	5.0	11.7	70	27	3
15	Moderate	22,000	9′ 30″	2′ 30″	Prolonged	Not done	Positive	90	4.5	9.3	52	45	3
16	Moderate	5,000	7′ 55″	2′ 23″	Prolonged	Not done	Negative	95	4.7	10.1	73	21	6
17	Severe	44,000	5′ 30″	4′	Prolonged	30**	Not done	88	4.5	9.6	49	49	2
18	Moderate	20,000	4′ 45″	5′	Normal	10	Positive	55	2.6	6.0	69	21	10
19	Mild	40,000	45′	4′ 45″	Normal	15	Not done	92	5.6	10.0	74	22	4
20	Mild	6,000	7′ 30″	4′ 30″	Not done	15	Not done	86	5.0	8.9	75	15	10

** Accuracy of this value open to question.

members of the spleen clinic, splenectomy is advised. In this connection, it may be said that, when possible, we prefer to remove the spleen during a remission of the disease. Immediate splenectomy is performed only as an emergency or semi-emergency measure.

In our experience, as we believe our results will show, splenectomy offers a greater hope of the complete arrest of purpura hemorrhagica than any other form of therapy. We have tried at various times vitamin C, sesame oil, progynon, oil of turpentine, ultraviolet light and multiple transfusions without noticeable success.

While we have not ourselves used snake venom, seven of our twenty-two operated cases had received this form of therapy before coming to the spleen clinic. In none did more than temporary relief result. Therefore we are led to believe that this form of therapy is of no permanent benefit in idiopathic thrombocytopenic purpura. In this connection, it may be of interest to note that all of the seven above-mentioned cases have been asymptomatic since splenectomy.

Immediately prior to operation, a transfusion needle is placed in the patient's arm. Throughout the operative procedure a compatible blood donor is held in readiness in an adjacent room. Platelet counts and capillary resistance determinations are performed before, during and shortly after removal of the spleen. It is not unusual for all bleeding to cease and for both the capillary resistance and platelet count to show appreciable increments immediately after removal of the spleen, but before the abdominal closure is completed. In the typical case, there is a cessation of the bleeding tendency and an abrupt rise in both of the latter values within the first twenty-four hours after operation. In most instances, the capillary resistance tends to rise more rapidly than the platelet count.

The postoperative course of these patients frequently tends to be stormy. Persistent distention and a remittent type of fever have been the two most common complications in our series of cases. The etiology of the fever has in all instances remained a mystery. We suspect that it may be due to a thrombophlebitis of the splenic vein. In at least two cases in which it occurred, it subsided abruptly following the administration of prontylin. It should be remembered, however, that in purpura this drug must be used with more than ordinary caution in view of its occasional detrimental action on the blood elements.

Despite the possible complications of the postoperative period and

the increased hazards encountered in operating upon individuals with this disease, we have no operative deaths to report in this group. In this connection, it cannot be emphasized too strongly that operation should only be attempted by an unusually well qualified surgeon.

Results

Up to October 1, 1938, forty-two patients with idiopathic thrombocytopenic purpura had been treated in the Spleen Clinic of the Presbyterian Hospital. Twenty-two of these individuals had undergone splenectomy. The remaining twenty had been treated conservatively.

One patient in the group undergoing splenectomy was operated upon too recently to warrant inclusion in the following tables. Consequently this case does not appear in Chart I which depicts the follow-up results in twenty-one operative cases.

In thirteen of these twenty-one cases, the disease appears to be permanently arrested. In five, because of some trivial incident in an otherwise perfect follow-up, it has been designated as markedly improved. Therefore the results in eighteen, or 85.7 per cent of this group may be said to have been eminently satisfactory.

Of the three remaining cases subjected to splenectomy, two have been improved, but by no means symptom-free, since operation, and one has died of the disease. This death occurred eighteen months after operation and was apparently caused by cerebral hemorrhage. Unfortunately, permission for an autopsy could not be obtained. The one other death in the operative group occurred in an individual whose purpura had been completely arrested for thirteen years. It was due to an unrelated, intercurrent disease.

The follow-up results on the twenty conservatively treated cases of idiopathic thrombocytopenic purpura are shown in Chart II. In only two of the twenty cases in this group does the disease appear to be completely arrested. In none can it be classified as markedly improved. Therefore, conservative therapy can be said to have been entirely successful in only 10 per cent of the group.

The follow-up reports on the eighteen patients remaining in this group may be summarized as follows: In four instances, the disease is still present, but has shown some degree of improvement; in thirteen instances, its initial severity remains unchanged; in one instance, it proved fatal, the patient having died in an acute flare-up of the disease.

IDIOPATHIC THROMBOCYTOPENIC PURPURA CESSATION OF ACTIVITY FOLLOWING SPLENECTOMY

Chart I

1. **Each circle represents six months in the clinical course.**
2. **The letter S indicates time of splenectomy during the entire period patient was followed.**
3. **Clear circles denote asymptomatic periods. Shaded circles denote periods with symptoms. Black circles denote death.**

At necropsy, death was found to have been due to cerebral hemorrhage.

It is therefore apparent that in these eighteen cases, which comprise 90 per cent of the conservatively treated group, the follow-up results were unsatisfactory.

If the operative and non-operative groups are compared (Table III), it will be seen that satisfactory results were obtained in 85.7 per cent of the operative and in only 10 per cent of the non-operative. On the other hand, splenectomy yielded unsatisfactory results in only 14.3

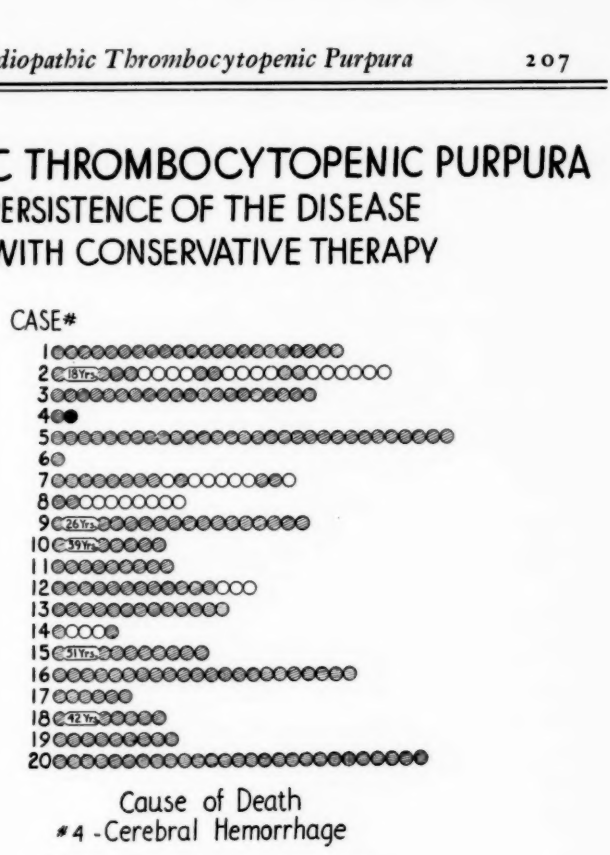

Chart II (refer to key under Chart I)

TABLE III

TOTALS IN THE TWO GROUPS

	With Operation	Without Operation
Satisfactory:		
Completely Arrested	13	2
Markedly Improved	5	0
	18 (85.7%)	2 (10.0%)
Unsatisfactory:		
Improved	2	4
Unimproved	0	13
Died*	1	1
	3 (14.3%)	18 (90.0%)

* Death due to purpura.

SPLENECTOMY IN SECONDARY PURPURA

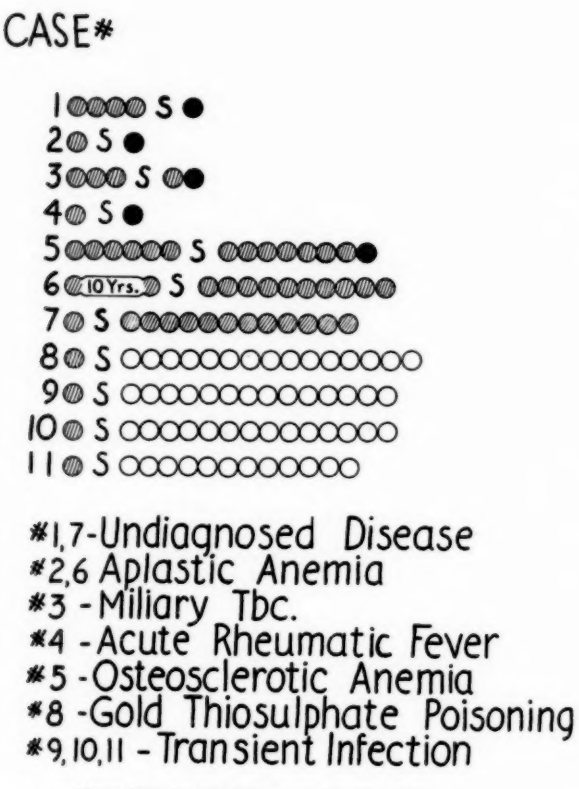

Chart III (refer to key under Chart I)

per cent of the cases so treated, while conservative therapy failed to produce satisfactory results in 90 per cent of the cases treated by this method. It is on this comparison that we base our contention that splenectomy is the most effective therapeutic measure at present employed in the treatment of idiopathic thrombocytopenic purpura. An essentially similar conclusion was reached by Wintrobe, Hanrahan and

Thomas[5] of Johns Hopkins in their paper on purpura hemorrhagica published in 1937.

Results of Splenectomy in Atypical Purpura

Chart III depicts the follow-up on eleven cases of atypical purpura which were subjected to splenectomy. In every instance the purpura proved, subsequent to operation, to be of the secondary, or symptomatic type.

Satisfactory results followed operation in only four instances. In one patient, the bleeding tendency has been improved slightly and in another, it does not seem to have been affected by operation. The remaining five patients are dead. One of these patients died before leaving the operating table, one succumbed to a postoperative complication and the remaining three died after leaving the hospital of diseases to which the purpura was apparently secondary.

In three of the four cases benefited by operation, the purpura was associated with acute, transient infection. In the fourth, it was apparently secondary to poisoning by gold thiosulphate. It seems not unlikely, therefore, that, had the associated etiologic factors in these four cases been eradicated prior to splenectomy, the bleeding tendency might have ceased without operative interference.

In the seven cases in which splenectomy was followed by unsatisfactory or fatal results, the purpura later proved to be associated with an aplastic anemia in two instances, with an undiagnosed disease process in two instances, and with an acute rheumatic carditis, a miliary tuberculosis and an osteosclerotic anemia in the remaining three respective instances.

This group of cases is presented to illustrate the diagnostic pitfalls, futile operations and high mortality so frequently encountered among the atypical purpuras.

Conclusions

1. In the vast majority of instances in which it is encountered, purpura represents a symptom-complex rather than a disease entity.

2. In children, thrombocytopenic purpura generally presents a different picture and a different problem from that observed in adults.

3. In adults, primary or idiopathic thrombocytopenic purpura is a relatively rare condition, occurring principally in women. Its course,

if untreated, is characterized by chronicity and recurrence.

4. The diagnosis of primary or idiopathic thrombocytopenic purpura should only be made after all of the etiologic factors known to be associated with the appearance of secondary purpura have been carefully excluded.

5. When performed by an experienced surgeon, splenectomy is the most effective form of therapy at present employed in the treatment of idiopathic thrombocytopenic purpura.

6. The removal of the spleen for the relief of hemorrhagic manifestations in cases of atypical or secondary purpura is emphatically contraindicated and is attended by a high incidence of disappointing and fatal results.

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PROCEEDINGS OF ACADEMY MEETINGS

STATED MEETINGS

NOVEMBER 3—*The New York Academy of Medicine.* Executive Session—a] Reading of the minutes; b] Report of Nominating Committee; c] Report on election of members. ¶ Papers of the evening (Graduate Fortnight)—a] The Wesley M. Carpenter lecture: Hemophilia, William H. Howell, Baltimore; b] Classification and treatment of purpura, Nathan Rosenthal, Associate in Medicine and Hematologist, The Mount Sinai Hospital.

NOVEMBER 17—*The Harvey Society (in affiliation with The New York Academy of Medicine).* The Second Harvey Lecture, The Significance of the Albumin Fraction of Serum, A. Ashley Weech, Associate Professor of Diseases of Children, College of Physicians and Surgeons.

DECEMBER 1—*The New York Academy of Medicine.* Executive Session—a] Reading of the minutes; b] Election of Academy Officers. ¶ Papers of the evening—Symposium on serum therapy in pneumonia—a] Present status of serum therapy, Russell L. Cecil, Professor of Clinical Medicine, Cornell University Medical College; b] Results with rabbit serum, Colin MacLeod (by invitation), Associate, The Rockefeller Institute for Medical Research; c] Program for meeting the pneumonia situation, Wheelan D. Sutliff (by invitation), Assistant Director, Pneumonia Control Division, Bureau of Laboratories, Department of Health; Discussion by Edward Tolstoi, Jesse G. M. Bullowa, Ralph S. Muckenfuss (by invitation).

DECEMBER 15—*The Harvey Society (in affiliation with The New York Academy of Medicine).* The third Harvey Lecture, Heat Loss from the Human Body, Eugene F. Du Bois, Professor of Medicine, Cornell University Medical College.

JANUARY 5—*The New York Academy of Medicine.* Executive Session—a] Reading of the minutes; b] Amendment to Constitution and By-Laws; c] Presentation of Diplomas. ¶ Presentation of annual reports (to be read by title) The Council, The Trustees, The Treasurer, Committees. ¶ Address of the retiring president, James Alexander Miller. ¶ Address of the incoming president, Malcolm Goodridge. ¶ Papers of the evening: Symposium on chronic gastritis—a] Recent advances in diagnosis by gastroscopy, Rudolf Schindler, Associate Professor of Medicine, The University of Chicago; b] Clinical aspects, Burrill B. Crohn, Associate in Medicine, The Mount Sinai Hospital. ¶ Report on election of members.

JANUARY 19—*The Harvey Society (in affiliation with The New York Academy of Medicine).* The fourth Harvey Lecture, "Proteins as Chemical Substances and Biological Components," Edwin J. Cohn, Professor of Biological Chemistry, Harvard Medical School.

SECTION MEETINGS

NOVEMBER 8—*Joint Meeting, Neurology and Psychiatry and the New York Neurological Society.* Papers of the Evening—a] Brief manic depressive cycle as an epileptic process, Richard M. Brickner, Albert Rosner (by invitation); Discussion by Nolan D. C. Lewis (by invitation); b] Pathological laughing and crying; a neuro-anatomical, neuro-physiological and psychological study, Charles Davison, Harold Kelman (by invitation); Discussion by Henry Alsop Riley, Nolan D. C. Lewis (by invitation); c] Some aspects of the biochemistry of alcoholic delirium, Herman Wortis (by invitation), S. Bernard Wortis, Paula Frank (by invitation), Norman Jolliffe (by invitation), Karl M. Bowman (by invitation); Discussion by Foster Kennedy.

Dermatology and Syphilology. This Section held no meeting because of conflict in date with the Graduate Fortnight.

Surgery. This Section held no meeting because of conflict in date with the Graduate Fortnight.

NOVEMBER 9—*Historical and Cultural Medicine.* Reading of the Minutes. ¶ Papers of the Evening—a] Sir Benjamin Ward Richardson's Hygeiopolis, and Jules Verne's Franceville, Ramsey Spillman; Discussion by Haven Emerson; b] The laryngeal cancer of Frederick the Third of Germany, George T. Pack; Discussion by James Ewing. ¶ General Discussion. ¶ Executive Session.

NOVEMBER 10—*Pediatrics.* On account of the meeting of the American Academy of Pediatrics at Rochester this month, there was no meeting of the Pediatric Section at the Academy.

NOVEMBER 15—*Medicine.* Reading of the Minutes. ¶ Papers of the Evening—a] The pathology and clinical manifestations of polyneuritis, I. S. Wechsler; Discussion by Foster Kennedy; b] The role of heavy metals and infections in polyneuritis, Leon H. Cornwall; Discussion by E. D. Friedman; c] Evaluation of vitamin B, (thiamine chloride) in the treatment of polyneuritis, Martin Vorhaus; Discussion by Norman Jolliffe.

NOVEMBER 16—*Otolaryngology.* Reading of the Minutes. ¶ Papers of the Evening: Symposium on otitic meningitis — a] Diagnosis—Bacteriological, A. A. Eggston; Differential, J. G. Dwyer; b] Avenues of infection, M. F. Jones; c] Operative treatment, Wesley Bowers; d] Chemotherapy, Emanuel Appelbaum. ¶ Discussion, Josephine B. Neal, Philip D. Kerrison.

NOVEMBER 16 — *Genito-Urinary Surgery.* Reading of the minutes. ¶ Papers of the evening — a] Endoscopic photography, Andrew Peterson (by invitation); b] Case report: aplastic kidneys, J. K. DeVries; c] New aspects in the treatment of vaginitis in children, Rose Andre (by invitation); d] Prevention of recurrent renal stone by calyceal resection, Francis Twinem; e] Treatment of postoperative and inoperable urological tuberculosis, Stanley Wang (by invitation); f] Urethrograms in infants and children, Paul M. Butterfield; g] Report of results of certain newer plastic operations in urology, Oswald S. Lowsley. ¶ General discussion.

NOVEMBER 18—*Orthopedic Surgery.* Instead of the regular meeting of Orthopedic Surgery at the Academy, the members met in Philadelphia at a joint meeting with the Philadelphia Orthopedic Club of Jefferson Medical College.

NOVEMBER 21—*Ophthalmology.* Instructional hour — Perimetry of chiasmal lesions, Ralph I. Lloyd. ¶ Slit lamp demonstration, Milton L. Berliner, Wendell L. Hughes, Girolamo Bonaccolto, Gordon M. Bruce. ¶ Reading of the minutes (8:30). ¶ Report of committees. ¶ Presentation of cases—a] Recurrence of ocular hypertension eighteen years after an Elliot trephining, Mark Schoenberg; b] A report on the use of sulfanilamide in the treatment of inclusion conjunctivitis, Phillips Thygeson (by invitation); c] Grenz ray in ophthalmology, Raymond Pfeiffer; Discussion by Gustave Bucky (by invitation). ¶ Papers of the evening —Interpretation of fundal changes associated with arterial hypertension, Walter I. Lillie (by invitation).

NOVEMBER 22—*Obstetrics and Gynecology.* Presentation of a case—A case of fibromyosis uteri, Frank Spielman (by invitation); Discussion by Samuel H. Geist. ¶ Papers of the evening—a] Sterilization in the female (Madlener technic), Erwin von Graff (by invitation); Discussion by Robert L. Dickinson, William H. Cary (by invitation), Mortimer N. Hyams; b] Pitfalls of version, Milton G. Potter, Buffalo (by invitation); Discussion by William E. Studdiford, William E. Caldwell. ¶ General discussion.

DECEMBER 2—*Surgery.* Reading of the minutes. ¶ Presentation of case reports—a] An abdominal reflex complicating anesthesia, Thomas H. Russell; Discussion by M. C. Peterson (by invitation); b] Toxic effects of carbon dioxide, Wallace B. Murphy (by invitation); Discussion by B. B. Lennon (by invitation); c] Untoward reaction with avertin, Henry W. Cave; Discussion by Lewis S. Booth (by invitation); d] Atelectasis complicating surgery, Louis Rene Kaufman; Discussion by Donald E. Brace; e] Anesthetic management during ruptured ectopic pregnancy, William E. Studdiford; Discussion by John Adriani (by invitation). ¶ Papers of the evening—a] Newer anesthetic agents, Paul M. Wood; Discussion by T. Drysdale Buchanan (by invitation); b] Newer anesthetic technics, E. A. Rovenstine; Discussion by T. Drysdale Buchanan (by invitation). ¶ General discussion.

DECEMBER 6—*Dermatology and Syphilology.* Reading of the minutes. ¶ Presentation of cases—a] Mt. Sinai Hospital; b] Miscellaneous cases. ¶ General discussion. ¶ Executive session.

DECEMBER 8—*Pediatrics.* Presentation of case, Brittain F. Payne. ¶ Papers of the evening—a] Normal development of the eye through infancy and childhood, Willis Knighton; b] The responsibility of the pediatricians in regard to children's eyes, Brittain F. Payne; c] Care of the eyes in acute contagious and infectious diseases, David Webster; d] Management of strabismus in infancy and childhood, Maynard Wheeler. ¶ Discussion, John Dunnington, Le Grand H. Hardy. ¶ Executive session.

DECEMBER 13—*Neurology and Psychiatry.* Reading of the minutes. ¶ Papers of the evening—a] Pathologic aspects of some forms of trauma to the brain and spinal cord, Lewis Stevenson; Discussion by Benjamin M. Vance, Milton Helpern; b] The place of Roentgen ray in the diagnosis of head injuries, Cornelius Dyke; Discussion by Leo M. Davidoff, Emanuel D. Friedman; c] A comparative study of the dehydrating action of dextrose, sucrose, sodium chloride, sorbitol and caffeine as determined by changes in the cerebrospinal fluid, Jefferson Browder (by invitation); Discussion by S. Bernard Wortis.

DECEMBER 16—*Orthopedic Surgery.* Reading of the minutes. ¶ Presentation of cases—a] Congenital subluxation of the fifth toe and its correction by a periosteocapsuloplasty operation, Leon Lantzounis; b] Cavernous angioma of the tibialis posticus muscle, Edgar D. Oppenheimer. ¶ Papers of the evening—a] Malignant giant cell tumor of the tibia; Case report, Samuel Kleinberg; Discussion by Henry L. Jaffe; b] The evolution of the spine fusion, M. Beckett Howorth; Discussion by Mather Cleveland; c] Three and one-half years experience with the corkscrew bolt and some points regarding its insertion, Robert K. Lippmann; Discussion by Philip Wilson. ¶ General discussion. ¶ Executive session.

DECEMBER 19—*Ophthalmology.* Instructional hour—Neurology of visual apparatus, George Arthur Blakeslee. ¶ Slit lamp demonstration, Milton L. Berliner, Wendell L. Hughes, Girolamo Bonaccolto, Gordon M. Bruce. ¶ Reading of the minutes (8:30). ¶ Presentation of cases—a] Gonioscopic apparatus, Donald W. Bogart (by invitation); b] A congenital type of endothelial dystrophy, Frederick H. Theodore (by invitation); c] A typical primary degeneration of the retina (retinitis pigmentosa sine pigmento). Report of three instances in a family of five children, Samuel P. Oast; d] Chorioretinitis of the macula. Report of a case with an extension from the macula area quiescent at least twelve years, Raymond Emory Meek; e] Bacterial synergism in lid necrosis. Report of a case, Isadore Givner. ¶ Paper of the evening—Fundus evidence of ocular injury, Arthur J. Bedell.

DECEMBER 20—*Joint Meeting, Medicine and the New York Diabetic Association.* Reading of the minutes. ¶ Papers of the evening—a] Present status of protamine

zinc insulin therapy, Herman Lande (by invitation); b] Protamine zinc insulin: A metabolic study of two severe cases of diabetes with comments on criteria for treatment, Edward Tolstoi; Discussion by Eugene F. Du Bois, George E. Anderson (by invitation); c] The management of diabetic acidosis and its accompanying "medical shock," H. Rawle Geyelin; Discussion by Louis Bauman.

DECEMBER 21 — *Genito-Urinary Surgery.* Reading of the minutes. ¶ New instrument—A cystoscope holder, Arthur A. Rosenthal. ¶ Case report—Two unusual cases of diverticuli of the urinary bladder, J. Sydney Ritter. ¶ Paper of the evening—The influence of accessory renal vessels in certain cases of hydronephrosis, Hugh J. Jewett, Baltimore (by invitation); Discussion by Augustus Harris, George F. Hoch, Thomas J. Kirwin. ¶ General discussion.

DECEMBER 21—*Otolaryngology.* Reading of the minutes. ¶ Papers of the evening—a] Deep infections of the neck, (1) Anatomy, John M. Lore, (2) Diagnosis and treatment, Henry B. Orton, (3) Discussion, Henry S. Dunning, J. Eastman Sheehan; b] Facial paralysis, (1) Nerve graft operation, Thomas G. Tickle, (2) Plastic reconstruction, Clarence R. Straatsma (by invitation), (3) Discussion by J. H. Jenney (by invitation).

DECEMBER 27—*Obstetrics and Gynecology.* Presentation of cases—a] Puerperal sepsis, following spontaneous delivery, sulfanilamide treatment, A. C. Posner (by invitation); b] Pre-eclampsia-twins, Meyer Rosensohn; c] Diabetes-Cesarean section, A. J. Fleischer (by invitation); d] Carcinoma developing in teratoma of ovary, (1) Clinical, I. Smiley (by invitation), (2) Pathological, L. Auster (by invitation); e] Granulosa cell tumor of ovary, H. B. Schoenberg; Discussion by F. R. Smith. ¶ Lantern slide demonstration—a] X-Ray visualization of soft tissues of pregnancy at term, Meyer Rosensohn, W. Snow; Discussion by J. R. Carty, Solomon Weintraub. ¶ Paper of the evening—a] Infectious diarrhea of newborn, Joseph Felsen; Discussion by Henry Heiman.

JANUARY 3—*Dermatology and Syphilology.* Presentation of cases—a] New York University College of Medicine and Bellevue Hospital; b] Miscellaneous cases. ¶ General discussion. ¶ Executive session.

JANUARY 6—*Surgery.* Reading of the minutes. ¶ Presentation of cases—a] Obstruction of the rectosigmoid by adhesions, Edwin B. Eckerson, Discussion by Edward J. Donovan; b] Colectomy and ileosigmoidostomy for multiple polyposis in a child 11 years of age, Irwin E. Siris, Discussion by John H. Morris; c] Fibrolipoma of the cecum, with ceco-colic intussusception and ulcerative colitis, Joseph H. Fobes, Discussion by Henry W. Cave; ¶ Papers of the evening—1. Malignancy of the colon simulating other diseases, A. Lightstone, Discussion by William C. White; 2. A simplification of the perineal operation, Jerome Lynch, Discussion by Vincent Hurley; 3. Multiple stage operations in surgery of the colon, Ralph Colp, Discussion by William F. MacFee. ¶ General discussion.

JANUARY 10—*Combined Meeting, Neurology and Psychiatry and the New York Neurological Society.* Papers of the evening—a] The treatment of depersonalization, Paul Schilder, Discussion by Manfred Sakel (by invitation), Louis Wender; b] A method for differentiating manic-depressive depressions from other depressions by means of parotid secretions, Edward I. Strongin, Leland E. Hinsie, Discussion by Karl M. Bowman (by invitation), Harold G. Wolff; c] Psychiatric disorders in fifty school teachers, James H. Wall (by invitation), Discussion by Robert B. McGraw, Leland E. Hinsie.

JANUARY 11—*Historical and Cultural Medicine.* Reading of the minutes. ¶ Papers

of the evening—a] Obstetrics at the New York Almshouse and Bellevue Hospital. Lantern slides, Claude Edwin Heaton, Discussion by William E. Caldwell; b] Obstetric forceps from the Chamberlens to the present. Lantern slides, Hervey C. Williamson, Discussion by B. P. Watson, Andrew A. Marchetti. ¶ General discussion. ¶ Executive session.

JANUARY 12—*Pediatrics.* Residents' night. Papers of the evening—a] New York University Medical College, "Subacute" pneumonia, Herbert Johnson, Bellevue Hospital (by invitation), Discussion opened by Edith M. Lincoln; b] New York University Medical College, Meningitis sympathica as an onset of Pott's disease, Lawrence Slobody, Metropolitan Hospital (by invitation), Anthony Maffia, Metropolitan Hospital (by invitation), Josephine B. Neal, Discussion opened by Reuel A. Benson; c] Cornell University Medical College, Dysostosis multiplex, James M. Hanks, New York Hospital (by invitation), Milton Berliner, Discussion opened by Samuel Z. Levine; d] College of Physicians and Surgeons, Treatment of pneumococcal infections with 2-sulfanilyl aminopyridine, Gilbert M. Jorgensen, Babies Hospital (by invitation), Discussion opened by Rustin McIntosh, David Rutstein (by invitation), Henry Christian (by invitation).

JANUARY 16—*Ophthalmology.* Instructional hour, Local anesthetics, Walter S. Atkinson. ¶ Slit lamp demonstration, Milton L. Berliner, Wendell L. Hughes, Girolamo Bonaccolto, Gordon M. Bruce. ¶ Reading of the minutes (8:30). ¶ Presentation of cases—a] Meningioma producing unilateral exophthalmos, James W. Smith; b] A calibrated nipple for contact lenses, Joseph I. Pascal; c] Keratitis in Hurler's disease, Milton L. Berliner; d] A rare tumor of the orbit. Color photomicrographs of biopsy material from orbit, sternum and gland from neck. Color photomicrographs showing blood picture, William McLean, Robert W. Ward (by invitation), William E. Youland (by invitation); e] Relation of vascular disease to retinitis. A new clinico-pathological study (with slides), S. A. Agatston.

JANUARY 17—*Medicine.* Reading of the minutes. ¶ Papers of the evening—a] Pneumonias other than pneumococcus pneumonia, Yale Kneeland (by invitation), Discussion by David D. Rutstein, Medical Consultant, Bureau of Pneumonia Control, Albany (by invitation); b] Preliminary report of the use of sulfanilamide pyridine (M & B 693) in the treatment of lobar pneumonia, 1. The absorption, acetylation and excretion of M & B 693, H. E. Stokinger (by invitation), 2. New York Hospital and Bellevue Hospital, Norman Plummer, Herbert Ensworth (by invitation), 3. Rockefeller Institute, Colin MacLeod (by invitation), 4. Presbyterian Hospital, C. A. Ragan, Jr., Crispin Cooke (by invitation), 5. Roosevelt Hospital, E. B. Sanford (by invitation), Discussion by A. R. Dochez, J. G. M. Bullowa.

JANUARY 18 — *Genito-Urinary Surgery.* Reading of the minutes. ¶ Case reports —a] Malignant hiccup as a complication of prostatic resection and cured by bilateral phrenicectomy, Meredith F. Campbell; b] Prostatectomy at age of one hundred and ten, J. Bayard Clark; c] Leiomyoma of the prostate with resumé of twenty cases, Morris Robert Keen (by invitation). ¶ Paper of the evening—Transurethral prostatic resection: Experience with 1,200 patients seventy years of age or more, Gershom J. Thompson, Rochester, Minnesota (by invitation), Discussion by Henry G. Bugbee and J. Sturdivant Read.

JANUARY 18—*Otolaryngology.* Reading of the minutes. ¶ Papers of the evening—Head trauma: diagnosis and treatment —a] Fractures involving the nose and face, J. D. Whitham (by invitation), Discussion by Joseph D. Kelly; b] Fractures of the skull involving the temporal bones, J. Winston Fowlkes, Discussion by Isidore Friesner; c] Osteomyelitis

of the skull, Joseph E. King, Discussion by E. Ross Faulkner; d] Relation of infection to pneumatization of the temporal bones, Luzius Ruedi, Zurich (by invitation). ¶ Executive session.

Orthopedic Surgery. No meeting of the Section of Orthopedic Surgery was held this month because of the Annual Meeting of the American Academy of Orthopedic Surgeons in Memphis, Tennessee.

JANUARY 24—*Obstetrics and Gynecology.* Program from Methodist Episcopal Hospital, Brooklyn. Arranged by Dr. Harvey B. Matthews. Presentation of case—Hysterectomy for placental polyp, Henry T. Hagstrom (by invitation). ¶ Papers of the evening—a] Intravenous dial in obstetrics, Report of 200 cases, Martin Z. Glynn (by invitation); b] Rupture of the uterus associated with pregnancy, Henry S. Acken, Jr. (by invitation); c] Ablatio placentae: A statistical study from the records of the Methodist Episcopal Hospital, George H. Davis (by invitation); d] Observations on postpartum hemorrhage, Harry W. Mayes (by invitation); e] Diagnosis and management of placenta previa, Ralph M. Beach (by invitation), Discussion by Howard C. Taylor, Jr., Harvey B. Matthews. ¶ General discussion.

AFFILIATED SOCIETIES

NOVEMBER 18—*New York Pathological Society (in affiliation with The New York Academy of Medicine).* Case reports—a] Adenocarcinoma of the jejunum, treated by entero-enterostomy, and complicated by intussusception, M. F. Wiener, Cumberland Hospital, 10 minutes; b] Suppurative thrombophlebitis of the azygos vein associated with lung abscess, Jacob Furth, New York Hospital, 10 minutes. ¶ Papers of the evening—a] Immunological studies with virus-induced and transplantable growths of rabbits, John Kidd, Rockefeller Institute for Medical Research (by invitation), 30 minutes; b] The use of the aspiration biopsy in the diagnosis of tumors, Fred Stewart, Memorial Hospital (by invitation), 30 minutes. ¶ Executive session.

NOVEMBER 21—*New York Roentgen Society.* Presentation of cases was omitted. ¶ Papers of the evening—Correlation of upright post-mortem chest teleroentgenograms with autopsy findings, with special reference to pulmonary embolism and infarction; Part 1 Pathological aspects, Benjamin Castleman, Boston (by invitation); Part 2 Roentgenological aspects, Aubrey O. Hampton, Boston (by invitation). ¶ Discussion opened by J. Burns Amberson, Jr. (by invitation), Paul Klemperer (by invitation). ¶ Executive session.

DECEMBER 19—*The New York Roentgen Society (in affiliation with The New York Academy of Medicine).* Presentation of cases. ¶ Paper of the evening—The role of small intestinal intubation in the treatment and diagnosis of intestinal obstruction, W. Osler Abbott (by invitation); Discussion by Octa Leigh (by invitation), Paul C. Swenson. ¶ Executive session.

DECEMBER 22—*New York Pathological Society (in affiliation with The New York Academy of Medicine).* Case reports (10 minutes each)—a] Cavernous transformation of the splenic vein simulating Banti's disease, Chester R. Brown, Amour F. Liber, Lincoln Hospital; b] Bronchiolar necrosis in the newborn, Sheldon A. Jacobson, Crown Heights Hospital. ¶ Papers of the evening (25 minutes each)—a] Osseous changes in cases of chronic renal insufficiency without hyperparathyroidism, A. M. Ginzler, H. L. Jaffe, Hospital for Joint Diseases; b] Histogenesis of mammary carcinoma. A study based on key-block sections of the whole gland, Harold Koppelman, New York University. ¶ Executive session.

JANUARY 16—*New York Roentgen Society (in affiliation with The New York Academy of Medicine).* Presentation of cases. ¶ Paper of the evening—Sialography—its technic and application in the diag-

nosis of diseases of the salivary glands, Alfred F. Hocker (by invitation), John V. Blady (by invitation). ¶ Discussion by Hayes E. Martin (by invitation). ¶ Executive session.

JANUARY 26—*New York Pathological Society (in affiliation with The New York Academy of Medicine).* Anniversary Meeting. Papers of the evening—a] Reaction of the nasal mucosa to infection with the virus of epidemic influenza, Thomas Francis, Jr., New York University (30 minutes); b] Carcinoma in frogs; its etiologic relation to a virus and its habits of growth *in vivo* and *in vitro* (illustrated by a motion picture), Balduin Lucke, Dept. of Pathology, University of Pennsylvania (45 minutes). ¶ Executive session. Election of officers.

DEATHS OF FELLOWS

BOETTIGER, CARL: 36-40 Bowne Street, Flushing, Long Island, New York; born in New York, in 1879; died in Forest Hills, New York, February 1, 1939; graduated in medicine from Cornell University Medical College in 1903; elected a Fellow of the Academy November 1, 1917.

Dr. Boettiger was physician to the St. John's Hospital, Long Island City, and the Queens General and Mary Immaculate Hospitals of Jamaica. He was a diplomate of the American Board of Internal Medicine, a Fellow of the American College of Surgeons, the American Medical Association, and a member of the Society of American Bacteriologists, the American Society of Clinical Pathologists, and the County and State Medical Societies.

JACHES, LEOPOLD: 100 East 94 Street, New York, N. Y.; born in Liban, Russia, December 28, 1873; died January 23, 1939; graduated in medicine from the College of Physicians and Surgeons in 1903; elected a Fellow of the Academy February 6, 1908.

Dr. Jaches was a member of the State and County Medical Societies, a Fellow of the American Medical Association and of the American College of Radiology, a member of the American Roentgen Ray Society, the Radiological Society of North America, the New York and the Philadelphia Roentgen Societies. He was also Radiologist to the Mt. Sinai Hospital, Consulting Roentgenologist to the Montefiore and the Norwalk (Conn.) Hospitals.

LECTURES ON OBSTETRICS

UNDER THE JOINT SPONSORSHIP OF

THE NEW YORK ACADEMY OF MEDICINE

AND THE

MEDICAL SOCIETY OF THE COUNTY OF NEW YORK

WEDNESDAY AFTERNOONS AT 4:30 O'CLOCK

2 East 103 Street

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March 1

The use of analgesics in labor

Thaddeus L. Montgomery, Philadelphia

March 8

Syphilis in pregnancy

Joseph N. Nathanson

March 15

Principles of hormone diagnosis and theories of endocrine therapy in pregnancy

Howard C. Taylor, Jr.

March 22

Management of pregnancy complicated by

(a) Tuberculosis

J. Burns Amberson, Jr.

(b) Heart disease

Edwin P. Maynard, Jr.

March 29

Recognition and management of abnormal presentations

Albert H. Aldridge

April 5

Sulfanilamide and other therapy in the treatment of post-abortal sepsis, post-partum sepsis and pyelitis

Edward G. Waters, Jersey City